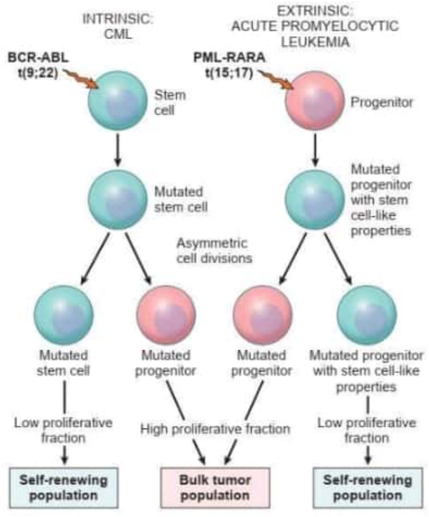
Hemato-Oncology

Oncometabolism

- Mutataions in Kreb's Cycle enzymes
- Lead to abnormal Glucse metabolism → forming abnormal metabolites→ **ONCOMETABOLITES**→ can Supress DNA methylation (of Protooncogenes) → cause Cancer.
- Eg: Isocitrate Dehydrogenase **IHD1** and **IDH 2** mutations lead to production of 2 Hydroxyglutarate(2HG) (which is an oncometabolite)
- 2HG → induces TET gene→ No DNA methylation → activation of Protooncogenes
- **Ivosidenib** and **Enasidenib** and IDH inhibitors



Cancer Stem Cells

GROWTH FACTOR RECEPTORS	GENES	TUMORS
EGF-receptor family	ERB-B1 (EGFR)	Adenocarcinoma Lung
	ERB-B2 (Her 2 Neu)	Breast, Stomach
FMS- like Tyrosine Kinase 3	FLT 3	Leukaemia
PDGF receptor	PDGFRB	Gliomas, Leukaemia
Receptor Tyrosine Kinase Ligand	KIT	GIST, Seminoma, AML

Growth factors	Tumours
PDGF	Astrocytoma
Fibroblast growth factor	Osteosarcoma, bladder
TGF-α	Astrocytoma
HGF	HCC, thyroid

Clinical Syndromes	Major Forms of Underlying Canc
Endocrinopathies	
Cushing syndrome	Small cell carcinoma of lung Pancreatic carcinoma Neural tumors
Syndrome of inappropriate antidiuretic hormone secretion	Small cell carcinoma of lung Intracranial neoplasms
Hypercalcemia	Squamous cell carcinoma of lung Breast carcinoma Renal carcinoma Adult T-cell leukemia/lymphoma
Hypoglycemia	Ovarian carcinoma Fibrosarcoma Other mesenchymal sarcomas
Polycythemia	Renal carcinoma Cerebellar hemangioma Hepatocellular carcinoma
Osteomalacia	Phosphaturic mesenchymal tumor
Nerve and Muscle Syndromes	
Myasthenia	Bronchogenic carcinoma Thymic neoplasms
Disorders of the central and peripheral nervous systems	Breast carcinoma
Dermatologic Disorders	
Acanthosis nigricans	Gastric carcinoma Lung carcinoma Uterine carcinoma
Dermatomyositis	Bronchogenic carcinoma Breast carcinoma
Osseous, Articular, and Soft Tissue Changes	
Hypertrophic osteoarthropathy and clubbing of the fingers	Bronchogenic carcinoma Thymic neoplasms
Vascular and Hematologic Changes	
Venous thrombosis (Trousseau phenomenon)	Pancreatic carcinoma Bronchogenic carcinoma Other cancers
Disseminated intravascular coagulation	Acute promyelocytic leukemia Prostatic carcinoma
Nonbacterial thrombotic endocarditis	Advanced cancers
Red cell aplasia	Thymic neoplasms

Cancer diagnosis & Screening

Tissue based

- **Biopsy**: Gold standard for most of malignancies
- **FNAC**: can be suggestive , primarily used as less invasive alternative
- **PAP Smears**: Screening for cervical Cancer
- **Liquid based Cytology**: Exfoliated cells are collected in fluid processed and observed under microscope (commonly used as an alternative for PAP smears)
- **Cytospin**: body fluids are centrifuged to observe tumour cells (e.g. Urine, Peritoneal / Pleural fluids, Broncho-alveolar lavages.
- **Exfoliative Cytology**: cells exfoliated are collected to check fortumour E.g: Sputum, Urine

- Tissue tumour markers on **IHC**
- **Microarray**: multiple gene mutations will be tested in single test Eg Breast carcinoma

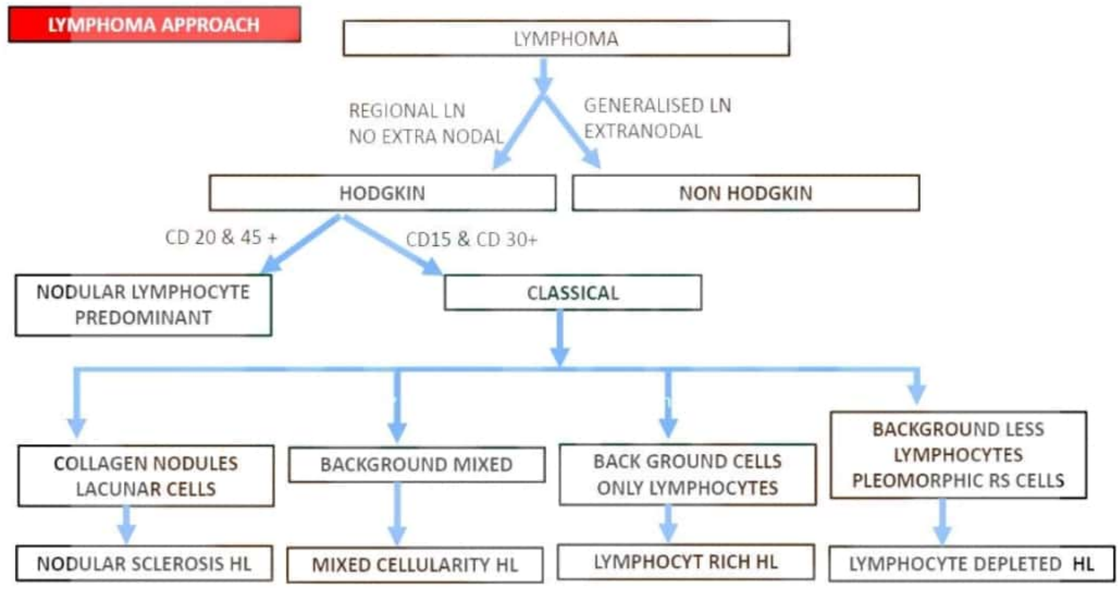
Blood based

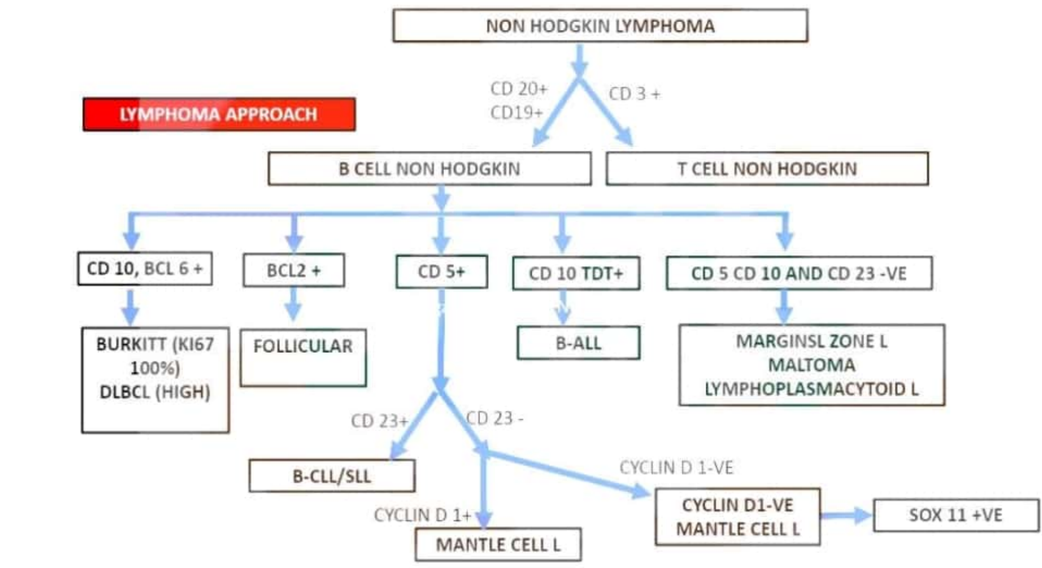
- Serum tumor markers

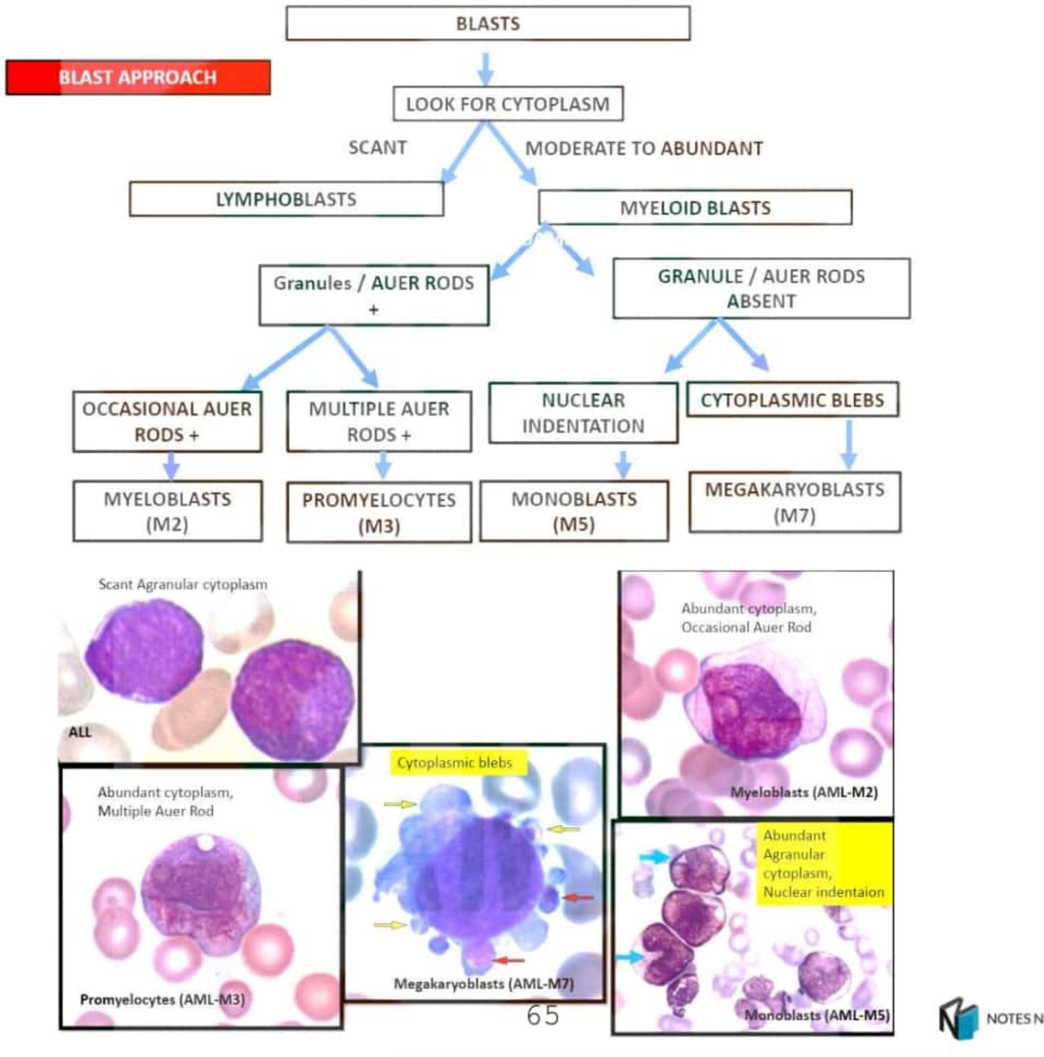
***Liquid biopsy** (important)*

- Blood / body fluid is collected to check tumor cells/ cell free tumor DNA for diagnosis

Cell-Free DNA Markers	
EGFR mutants in serum	Lung cancer
TP53, APC, RAS mutants in stool and serum	Colon cancer
TP53, RAS mutants in stool and serum	Pancreatic cancer
TP53, RAS mutants in sputum and serum	Lung cancer
TP53 mutants in urine	Bladder cancer







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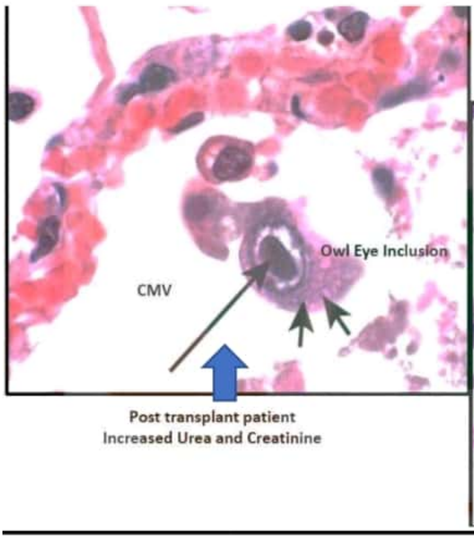
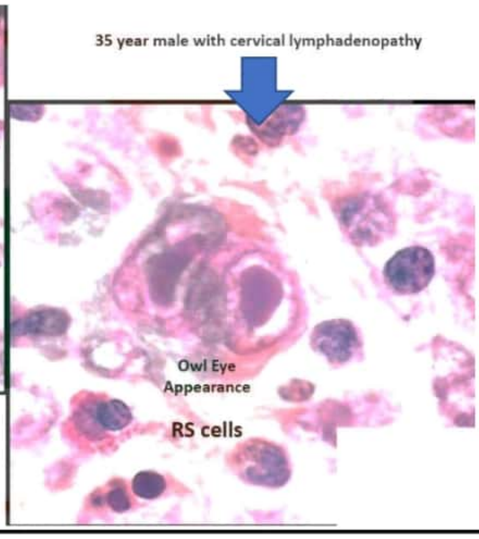
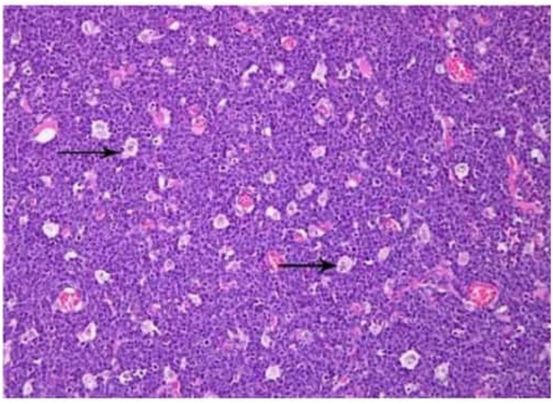


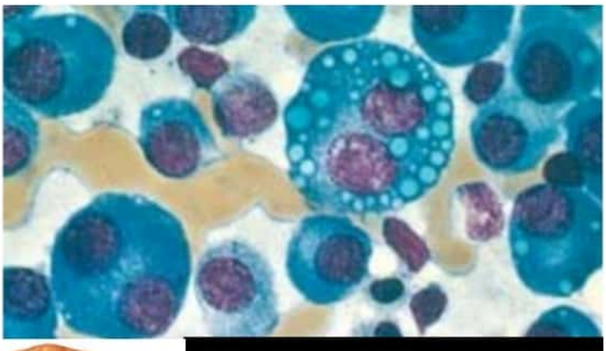


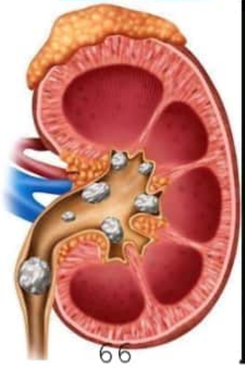


Diagnosis: **Burkitt's Lymphoma**
Neoplastic cells form the 'Blue Sky'
Arrows: Macrophages ('Stars')
Starry sky pattern

Diagnosis: **Multiple Myeloma**

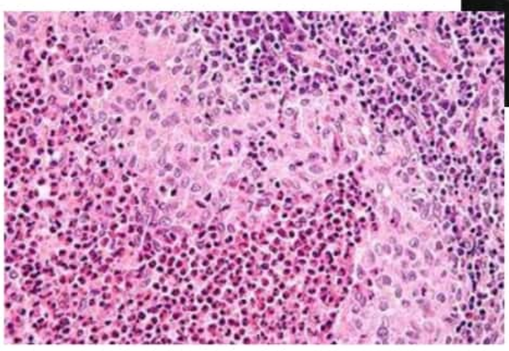
- Elderly age group
- Mott Cells in BM Aspirate
- Renal Stones
- Multiple "Punched Out" lytic lesions in the skull on Xray

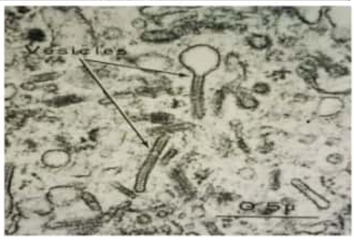


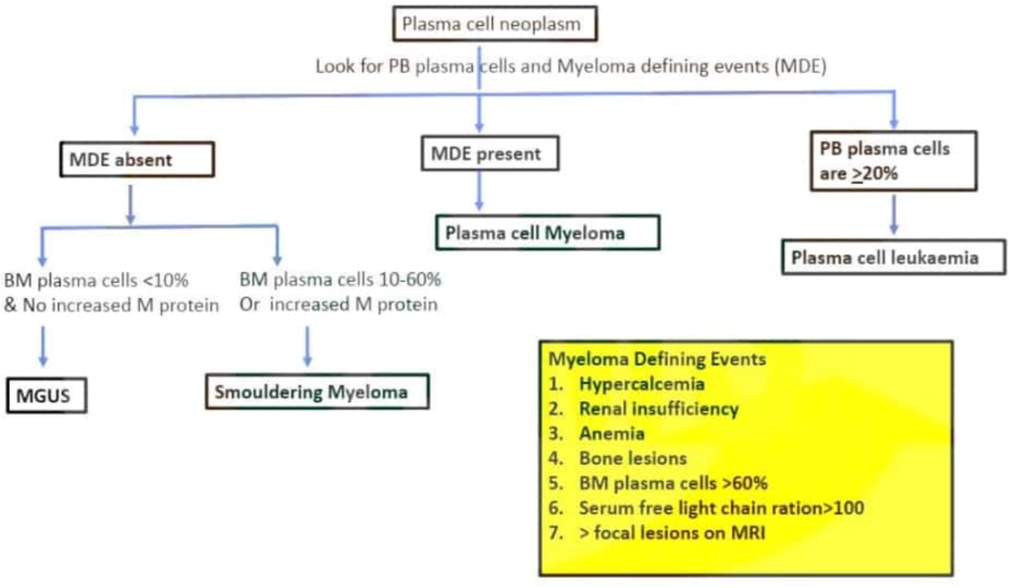


Diagnosis: **Langerhan cell histiocytosis**

- Age group: 2-year child
- Birbeck granules (Tennis racquet appr in EM)
- Eosinophilic granuloma with macrophages/histiocytes
- Skull Xray: Lytic Lesion



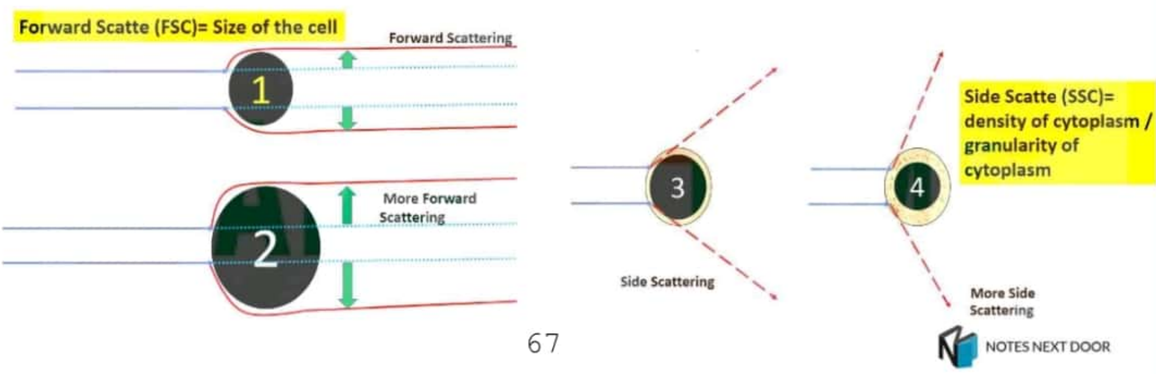


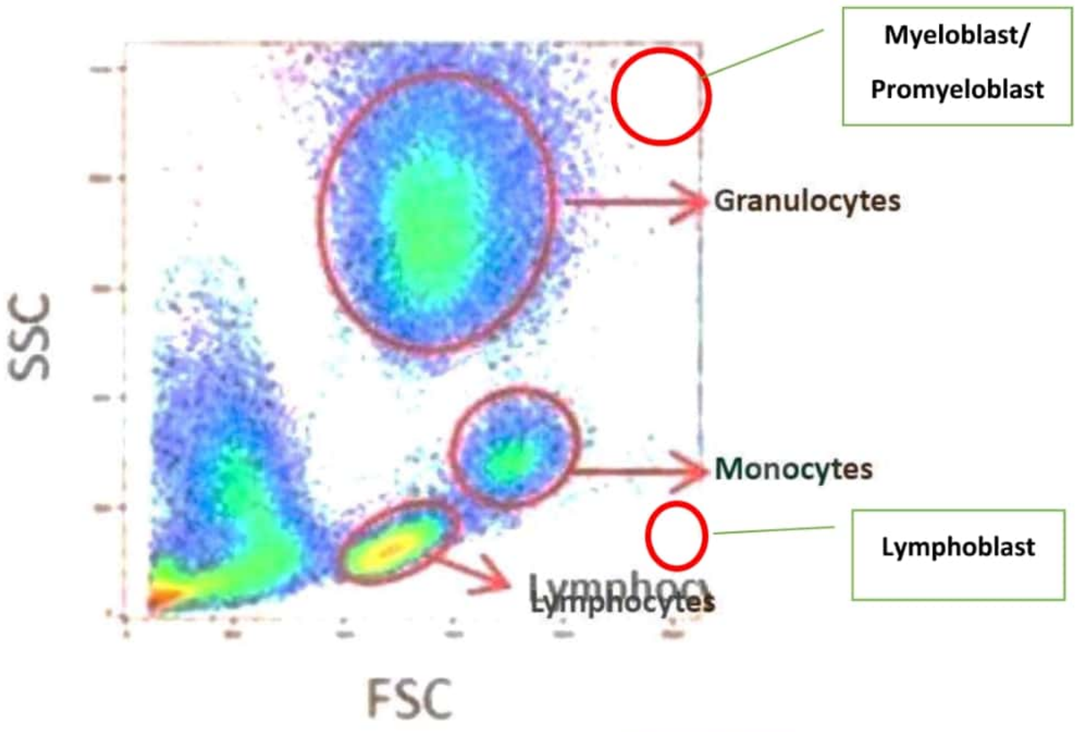

Plasma cell neoplasm
Look for PB plasma cells and Myeloma defining events (MDE)
MDE absent
MDE present
PB plasma cells are ≥20%
Plasma cell Myeloma
Plasma cell leukaemia
BM plasma cells <10% & No increased M protein
BM plasma cells 10-60% Or increased M protein
MGUS
Smouldering Myeloma

Myeloma Defining Events

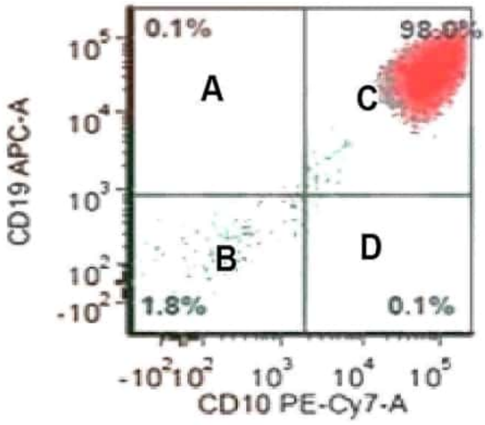
1. Hypercalcemia
2. Renal insufficiency
3. Anemia
4. Bone lesions
5. BM plasma cells >60%
6. Serum free light chain ration>100
7. > focal lesions on MRI

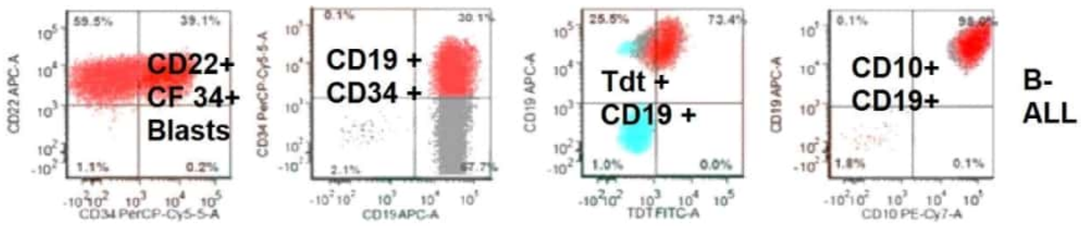
Flowcytometry





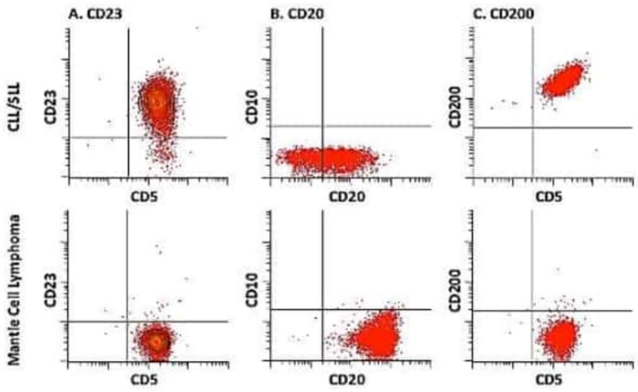
A= CD19 +ve , CD10 -ve
B= CD19 -ve , CD10 -ve
C= CD19 +ve , CD10 +ve
D= CD19 -ve , CD 10 +ve

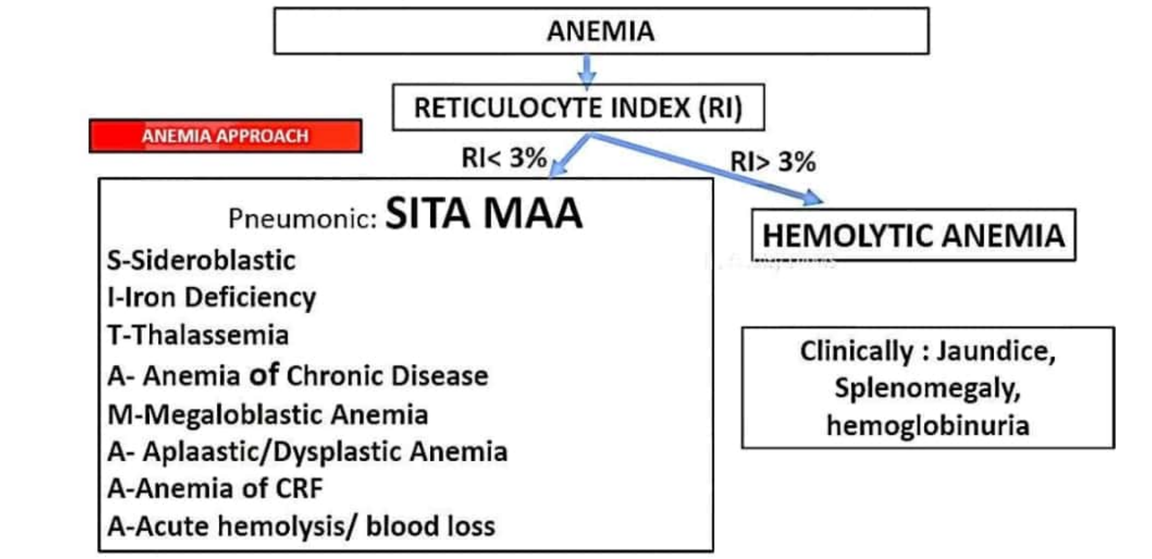


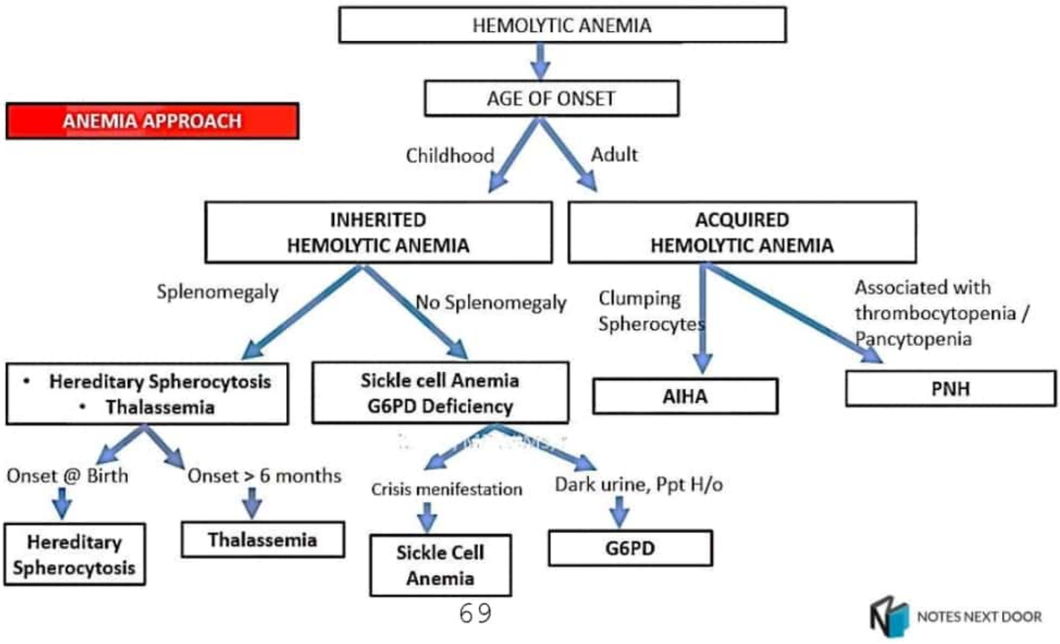


B cell Lymphoma with CD5 + & CD23+
Dx: **B-CLL/SLL**

Mantle Cell Lymphoma







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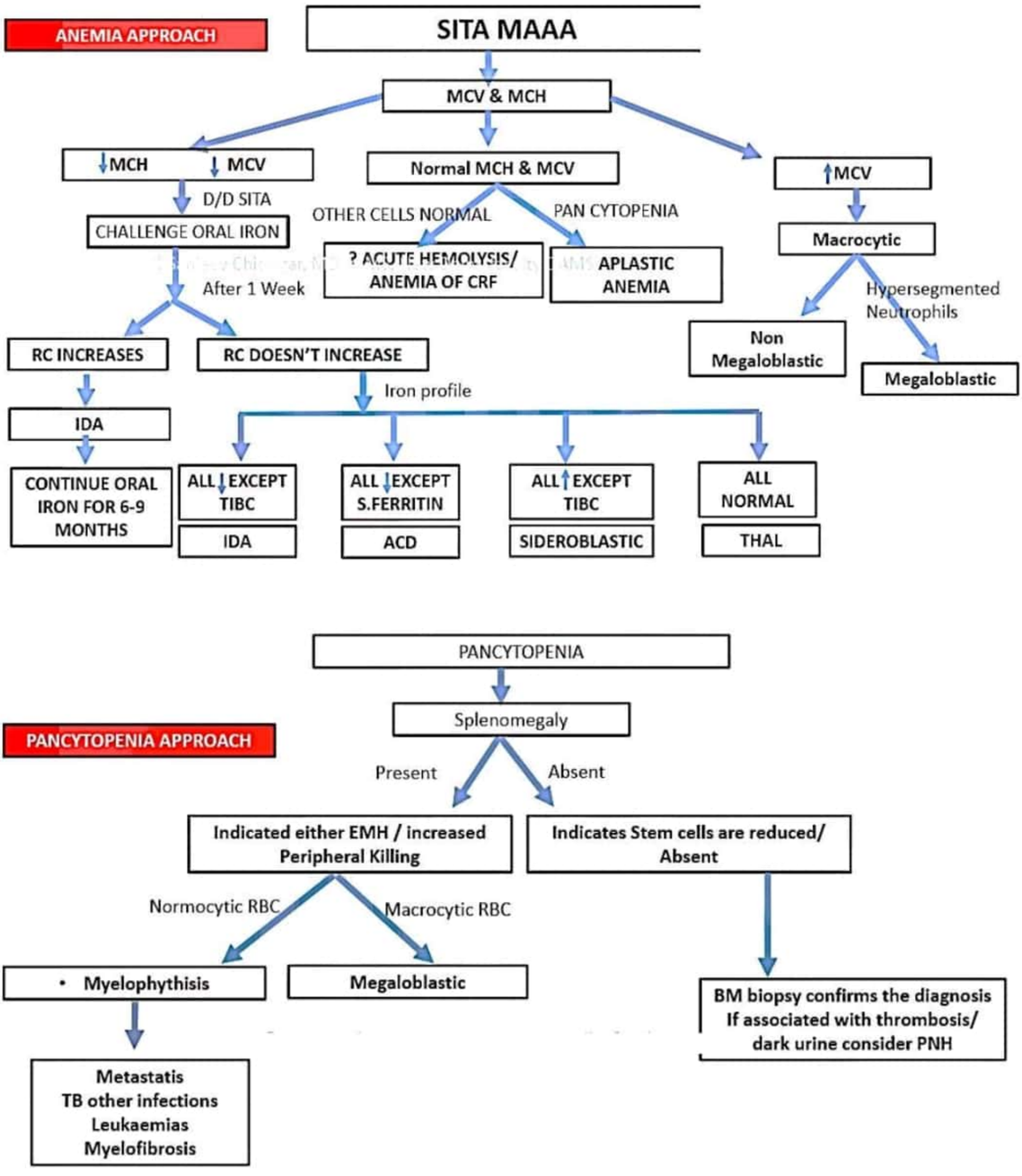
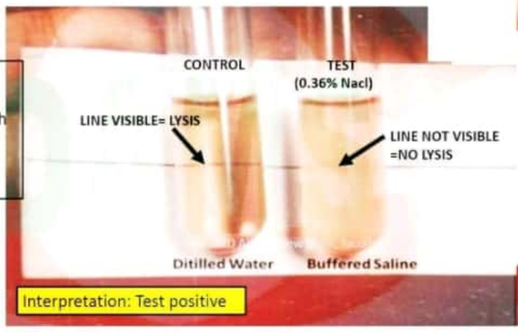
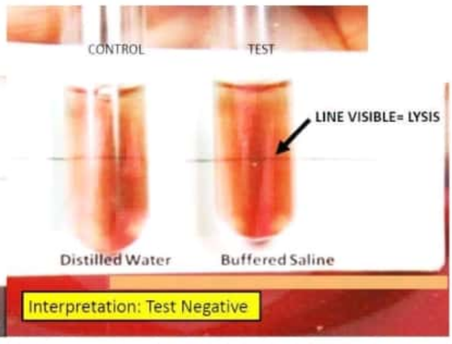

ANEMIA APPROACH
SITA MAAA
MCV & MCH
↓MCH ↓ MCV
Normal MCH & MCV
↑MCV
D/D SITA
CHALLENGE ORAL IRON
OTHER CELLS NORMAL
PAN CYTOPENIA
? ACUTE HEMOLYSIS/ ANEMIA OF CRF
APLASTIC ANEMIA
Macrocytic
After 1 Week
Hypersegmented Neutrophils
RC INCREASES
RC DOESN'T INCREASE
Non Megaloblastic
Megaloblastic
Iron profile
IDA
CONTINUE ORAL IRON FOR 6-9 MONTHS
ALL ↓ EXCEPT TIBC
IDA
ALL ↓ EXCEPT S.FERRITIN
ACD
ALL ↑ EXCEPT TIBC
SIDEROBLASTIC
ALL NORMAL
THAL

PANCYTOPENIA
Splenomegaly
PANCYTOPENIA APPROACH
Present
Absent
Indicated either EMH / increased Peripheral Killing
Indicates Stem cells are reduced/ Absent
Normocytic RBC
Macrocytic RBC
• Myelophthisis
Megaloblastic
BM biopsy confirms the diagnosis If associated with thrombosis/ dark urine consider PNH
Metastatis
TB other infections
Leukaemias
Myelofibrosis

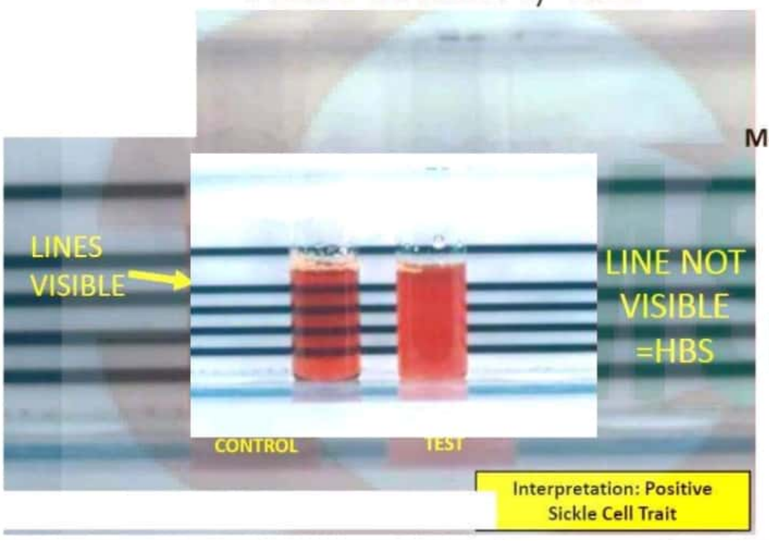
NESTROF test

Principle:
Thalassemia (minor) have Microcytes which do not undergo Lysis even in hypotonic Saline





Sickle solubility test

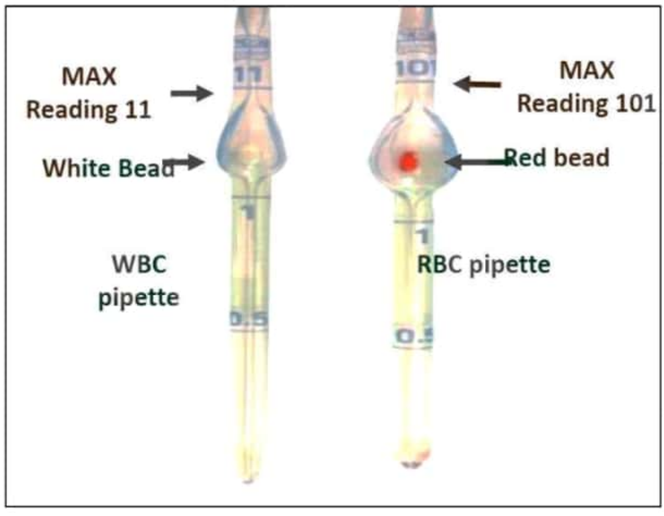


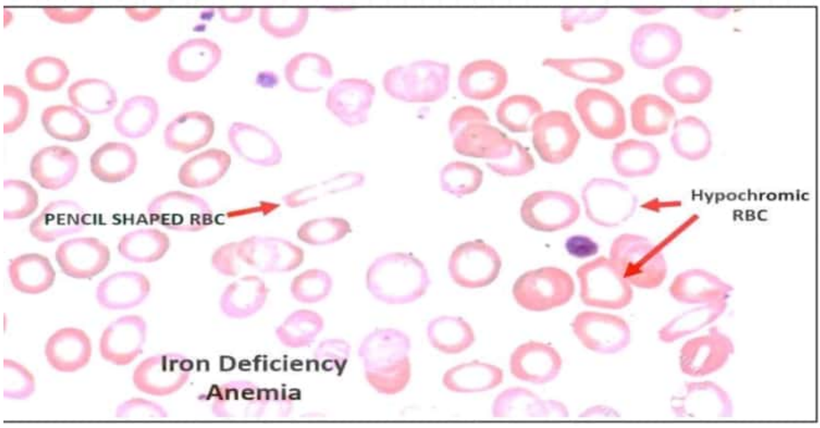
Blood

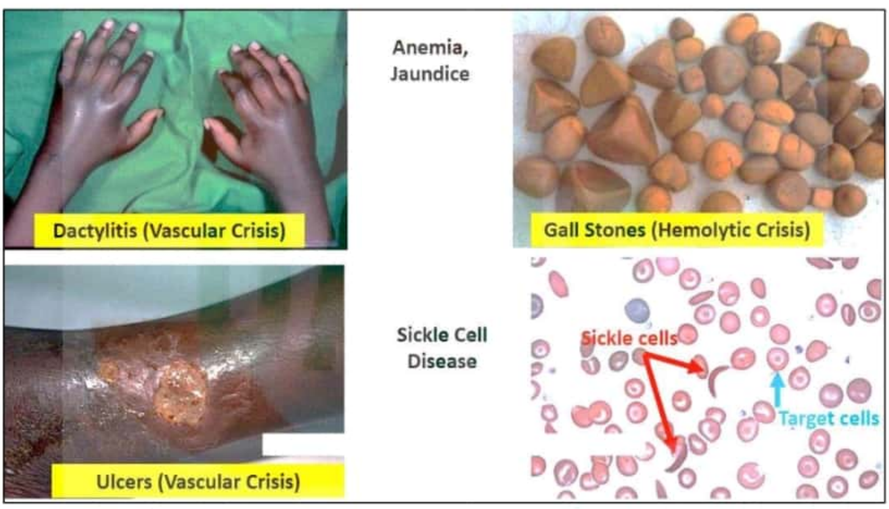
Sodium Metabisulpite

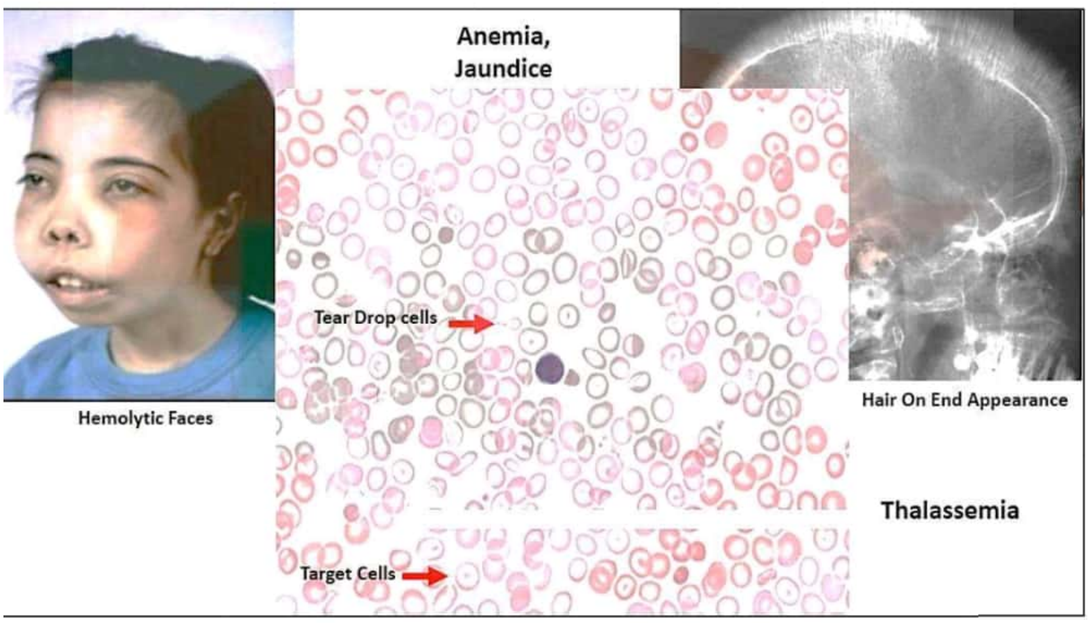
HbS becomes into insoluble Polymer

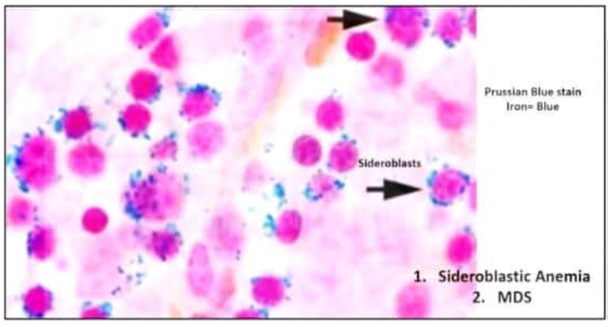
Lines not Visible

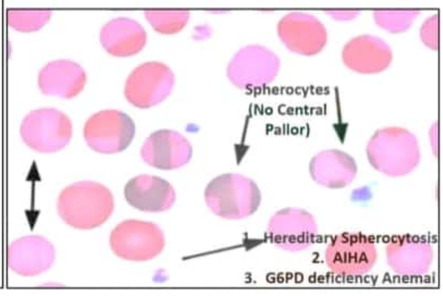


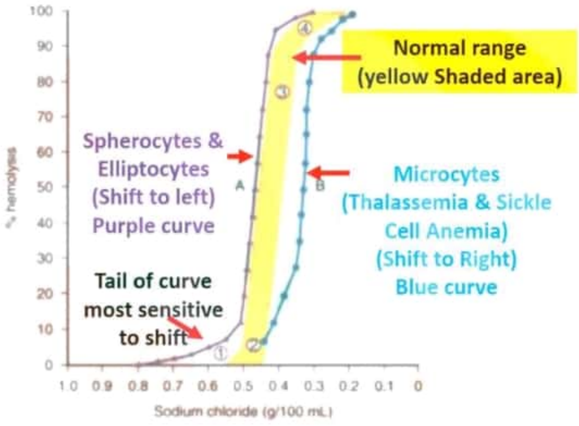

PENCIL SHAPED RBC
Hypochromic RBC
Iron Deficiency Anemia


Dactylitis (Vascular Crisis)
Anemia, Jaundice
Gall Stones (Hemolytic Crisis)
Ulcers (Vascular Crisis)
Sickle Cell Disease
Sickle cells
Target cells

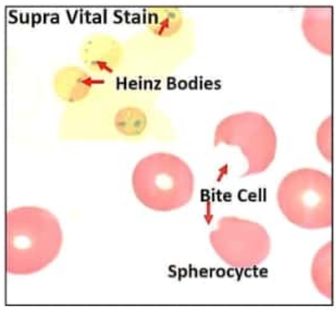

Anemia, Jaundice
Hemolytic Faces
Tear Drop cells
Hair On End Appearance
Thalassemia
Target Cells

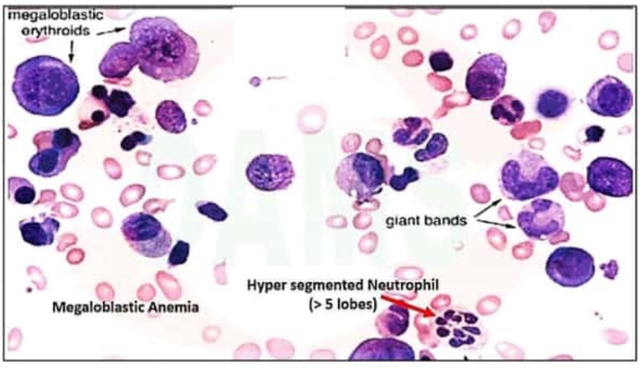


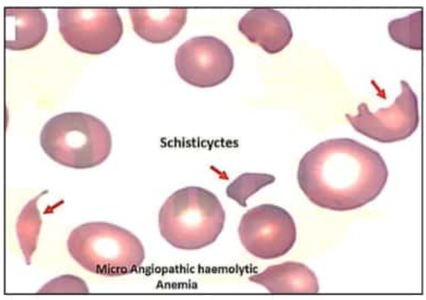




Heparin is the anticoagulant of choice here

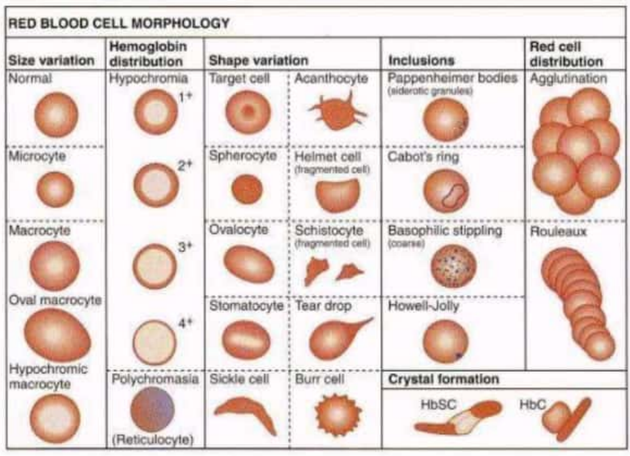






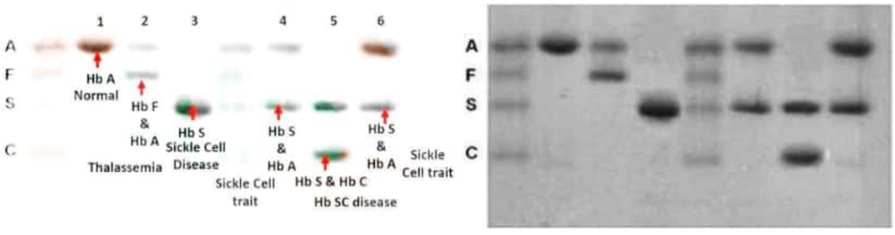
Shape	Appearance	Causes
Spherocyte	No central pallor	Hereditary spherocytosis, AIHA, G6PD deficiency anemia
Acanthocyte	irregular spicules on their surfaces	Abetalipoproteinemia

Elliptocytosis	Elliptical shape	Hereditary elliptocytosis
Echinocyte (burr cell)	Undulation on surface	Uremia and anaemia of CRF
Schistocyte (helmet cell)	Irregular shape	Microangiopathic hemolytic anemia (MAHA)
Bite cell	Pinched off cytoplasm	G6PD deficiency anemia
Tear drop cells (dacrocytes)	RbC LIKE ATEAR DROP	Thalassemia, myelofibrosis
Sickle cells	Shaped like half moon	Sickle cell anemia, Hb Sc disease

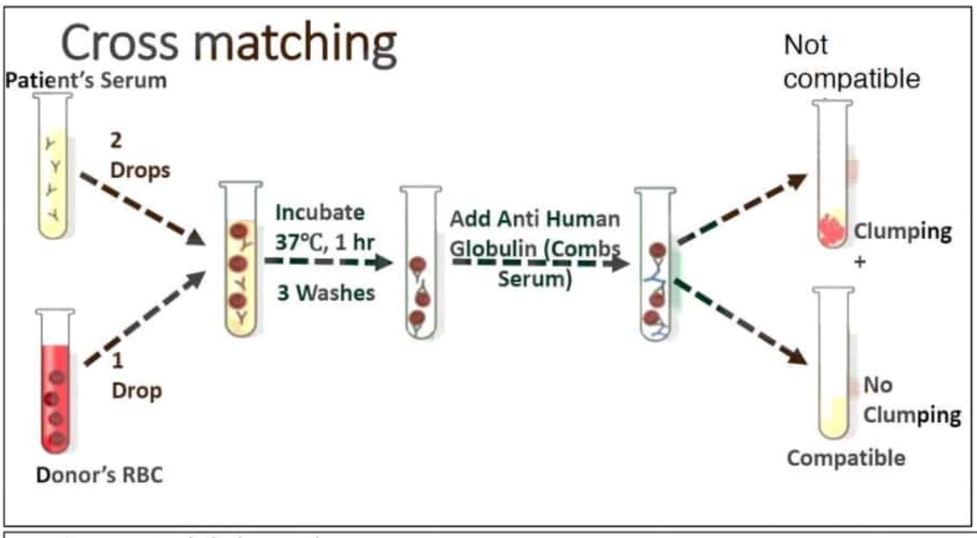


Name / content	Appearance	Conditions
Target cells	Hb in central pallor	Thalassemia (major, minor & intermedia), HbSC disease, liver disease, IDA, Hyposplenism
Basophilic stippling (composed of RNA/ ribosome)	Multiple granules in cytoplasm	Thalassemia, MDS, heavy metal poisoning, sideroblastic anemia, megaloblastic anemia
Howel jolly bodies (DNA)	Single dark blue like pyknotic nuclei	Hemolytic anemia, megaloblastic anemia, hyposplenic states

Cabot rings (mitotic spindle)	Ring or figure of 8	Megaloblastic anemia, hemolytic anemia
HbH inclusions	Golf ball appearance	Thalassemia, erythroleukemia
Heinz bodies (denatured Hemoglobin)	Dot like structure seen on supravital stains	G6PD deficiency anemia
Pappenheimer bodies (iron)	Large blue like howel jolly bodies but more than one	Sideroblastic anemia
Sideroblasts (iron)	Multiple granules of iron (seen after Prussian blue stain)	Sideroblastic anemia, MDS



Order & Color	Contents	Uses	Special Instructions
1. Culture bottle	Media	Culture	Aerobic first later Anaerobic
2. Sky Blue	Sodium Citrate (1:9)	Coagulation	Fill up to the marking provided 4 inversions
3. Black	Sodium Citrate (1:4)	ESR	
4. Red (serum tube)	Powdered glass	Serology, indirect coombs test, Cross matching	6 inversions
5.Gold	Polymer gel with powdered glass	Serology, immunology, B12, folate, Iron levels	6 inversions
6.Green	Na/ Li Heparin	Arterial blood gas, Chemistry, Lithium levels (Na Heparin)	8 inversions
7. Levander	EDTA	CBC,Direct Coombs test, Blood group, Lymphocytes for Markers	8 inversions
8 Yellow	Acid Citrate Dextrose	HLA, Paternity tests, DNA studies	
9. Gray	Oxalate + Na Fluride	Blood Glucose, Alcohol, lactic acid	

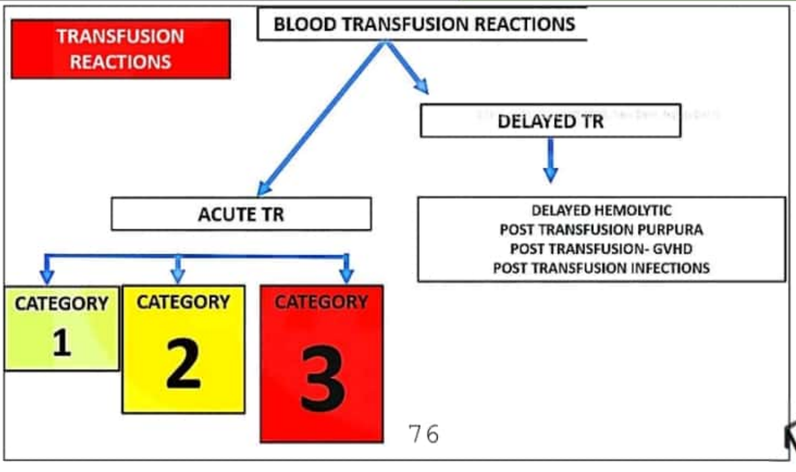


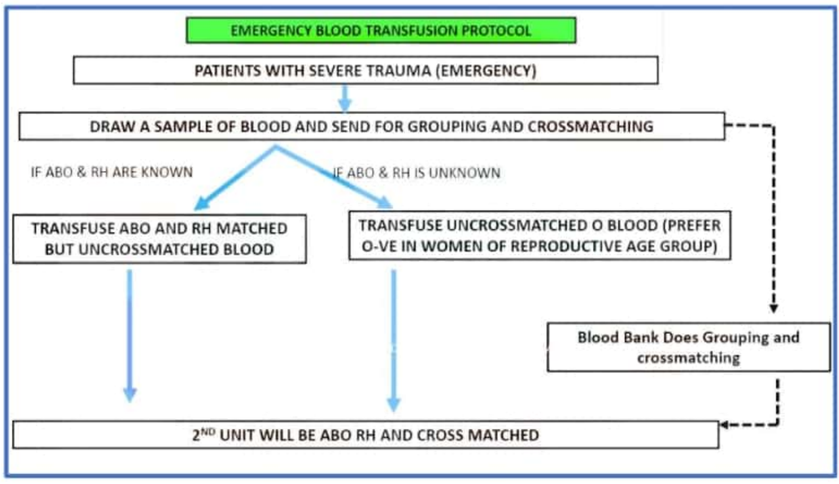
Special blood components

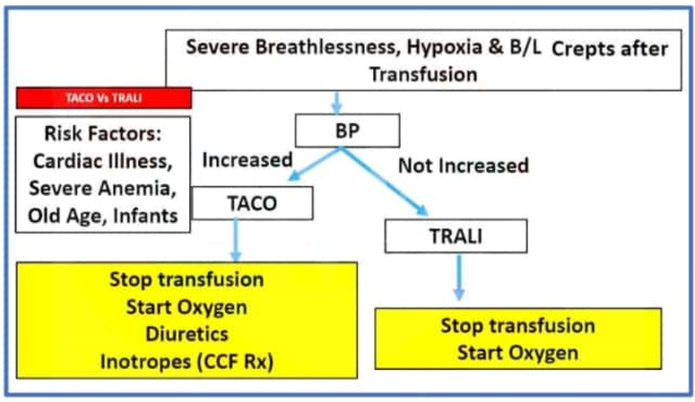
Saline washed RBC
- Uses: patients with allergic / anaphylactic reaction
  Eg: IgA deficiency, Jobs Syndrome, Wiskott Aldrich Syndrome
- Shelf life only 24 hrs

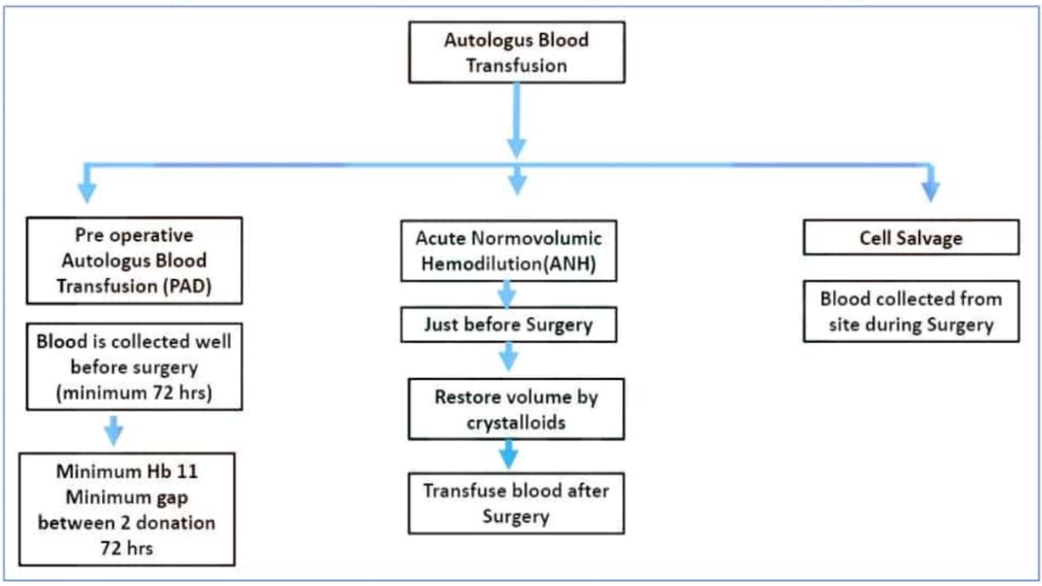
Irradiated blood
- Shelf life 14 days
- Uses :
  Relative donors blood
  In immunocompromised patient
  Intra uterine exchange transfusion

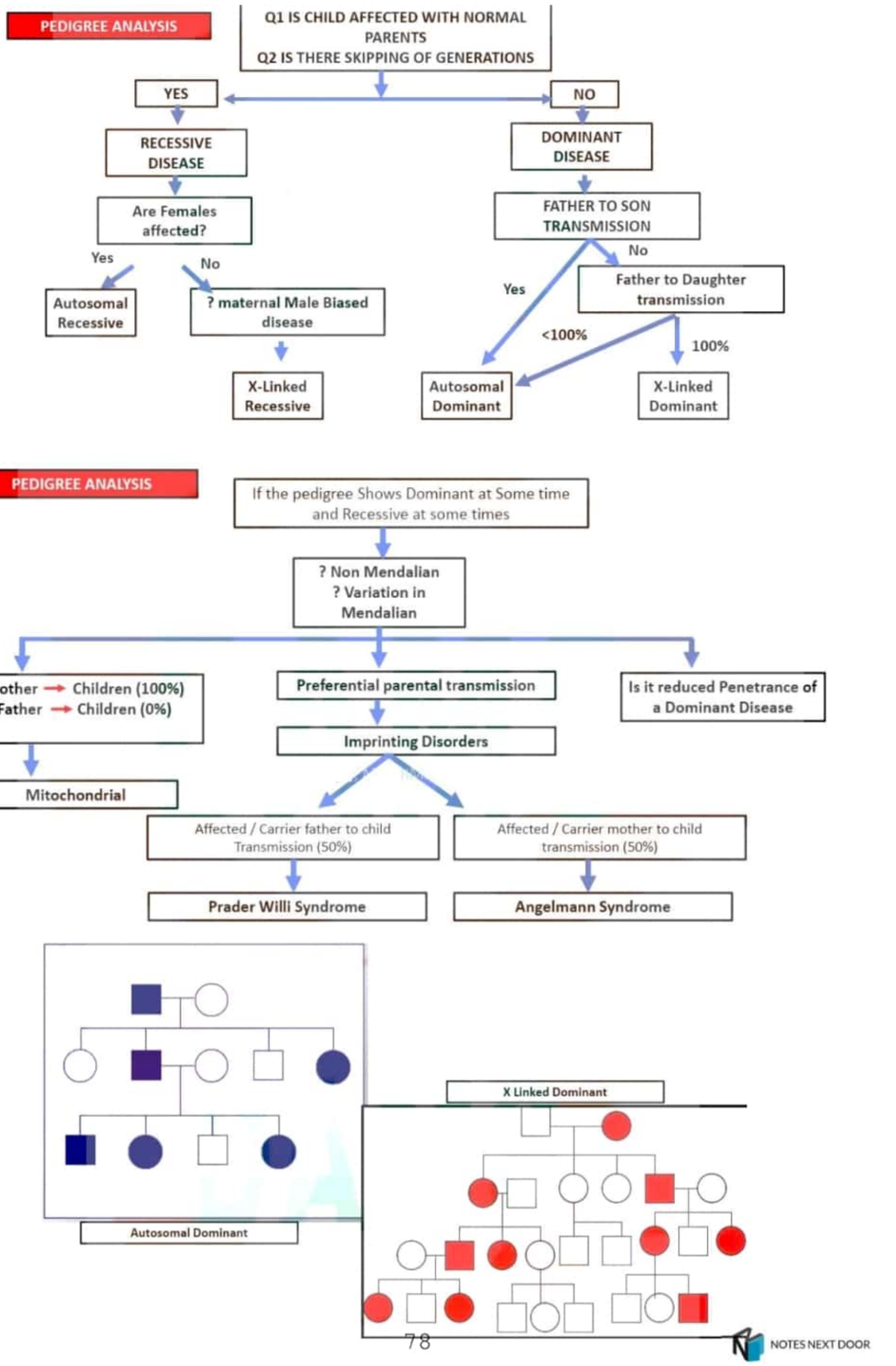
Fresh blood
- Blood collected < 7 days
- Indications: renal and hepatic dysfunction
  Intravascular hemolysis
  Burns
  Massive Blood
  Intrauterine exchange transfusion
- More chance of infection:
  Malaria (doesn't survive > 7 hrs) and Syphillis (doesn't survive >96 hrs in stored blood)
  CMV and HTLV (leucocytes)

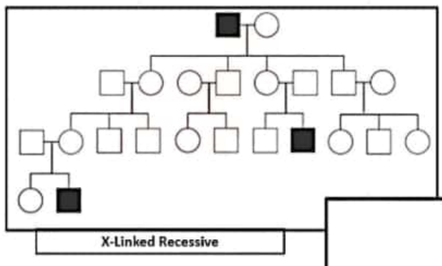



EMERGENCY BLOOD TRANSFUSION PROTOCOL
PATIENTS WITH SEVERE TRAUMA (EMERGENCY)
DRAW A SAMPLE OF BLOOD AND SEND FOR GROUPING AND CROSSMATCHING
IF ABO & RH ARE KNOWN
IF ABO & RH IS UNKNOWN
TRANSFUSE ABO AND RH MATCHED BUT UNCROSSMATCHED BLOOD
TRANSFUSE UNCROSSMATCHED O BLOOD (PREFER O-VE IN WOMEN OF REPRODUCTIVE AGE GROUP)
Blood Bank Does Grouping and crossmatching
2ND UNIT WILL BE ABO RH AND CROSS MATCHED

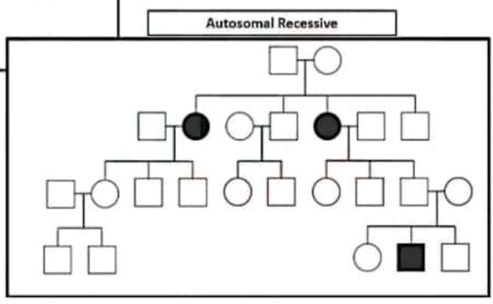

Severe Breathlessness, Hypoxia & B/L Crepts after Transfusion
TACO Vs TRALI
Risk Factors: Cardiac Illness, Severe Anemia, Old Age, Infants
BP
Increased
Not Increased
TACO
TRALI
Stop transfusion
Start Oxygen
Diuretics
Inotropes (CCF Rx)
Stop transfusion
Start Oxygen


Autologus Blood Transfusion
Pre operative Autologus Blood Transfusion (PAD)
Blood is collected well before surgery (minimum 72 hrs)
Minimum Hb 11
Minimum gap between 2 donation 72 hrs
Acute Normovolumic Hemodilution(ANH)
Just before Surgery
Restore volume by crystalloids
Transfuse blood after Surgery
Cell Salvage
Blood collected from site during Surgery

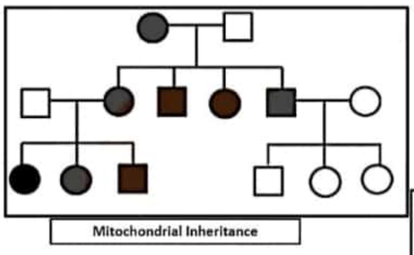

PEDIGREE ANALYSIS
Q1 IS CHILD AFFECTED WITH NORMAL PARENTS
Q2 IS THERE SKIPPING OF GENERATIONS
YES
NO
RECESSIVE DISEASE
DOMINANT DISEASE
Are Females affected?
FATHER TO SON TRANSMISSION
Yes
No
Autosomal Recessive
? maternal Male Biased disease
Yes
No
Father to Daughter transmission
<100%
100%
X-Linked Recessive
Autosomal Dominant
X-Linked Dominant
PEDIGREE ANALYSIS
If the pedigree Shows Dominant at Some time and Recessive at some times
? Non Mendalian
? Variation in Mendalian
Mother → Children (100%)
Father → Children (0%)
Preferential parental transmission
Is it reduced Penetrance of a Dominant Disease
Imprinting Disorders
Mitochondrial
Affected / Carrier father to child Transmission (50%)
Affected / Carrier mother to child transmission (50%)
Prader Willi Syndrome
Angelmann Syndrome
X Linked Dominant
Autosomal Dominant
78
NOTES NEXT DOOR



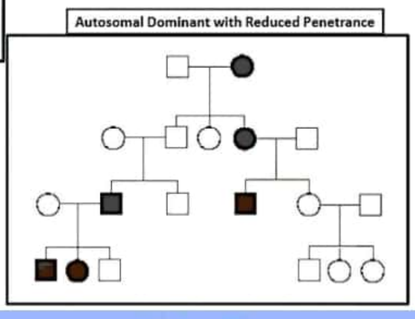
X-Linked Recessive



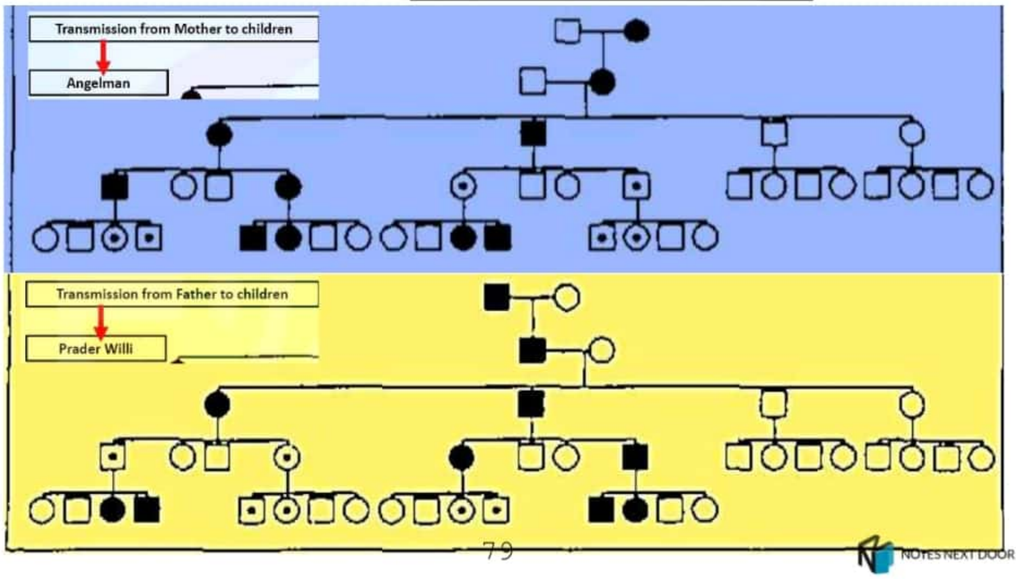
Autosomal Recessive



Mitochondrial Inheritance



Autosomal Dominant with Reduced Penetrance

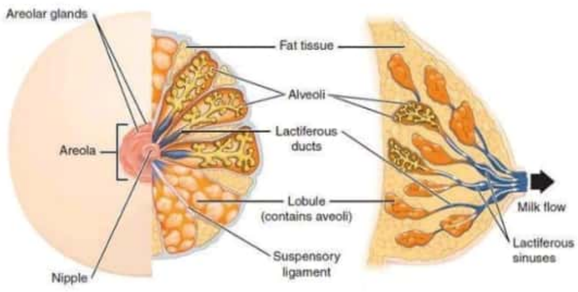


NEOPLASMS INTEGRATED

BREAST

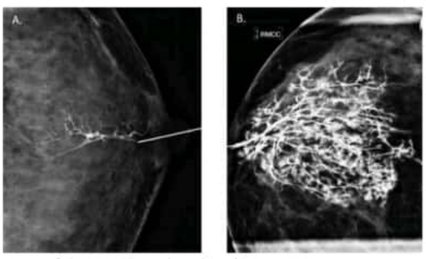
Ductal System Anatomy

- Anatomical Unit: Lobules (Consists of stroma and acinus)
- Functional Unit: Terminal duct lobular unit (TDLU)
- TDLU is the collective term for lobule with the terminal ductule



Nipple Discharge

- M/C/C of discharge:
  - Overall: Duct Ectasia
  - Bloody discharge: Intraductal papilloma
  - Greenish-black: Duct Ectasia
  - Purulent: Mastitis
  - Milk: Lactation
- Investigation: Ductography
  - Occasionally used to evaluate the surgically significant nipple discharge
  - Ductography is performed by cannulation of an individual duct orifice and injection of contrast material.
  - This ductography opacifies the entire ductal tree from the retroareolar duct to the lobules at the end of the tree
  - It also demonstrates the functional independence of each duct system.
  - There is no cross communication between independent duct systems.



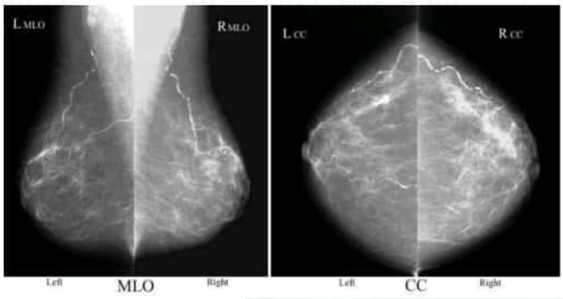
PAGET'S Disease

- Skin manifestation of underlying carcinoma
- Mostly a/w DCIS >> Invasive Ductal Ca
- Tumor Marker: CEA, Mucicarmine, Mammoglobin
- Management: Simple mastectomy or BCS
- Requires MRM if proven for invasive Ca

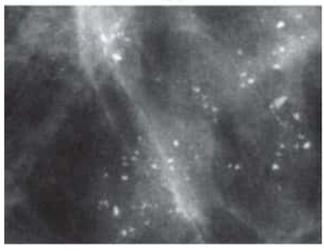
PEU' D ORANGE

- Represents the subcutaneous lymphatic edema
- There is extensive dermal invasion of tumor into the lymphatic channels
- Considered as T4b
- Represents LABC

MAMMOGRAPHY

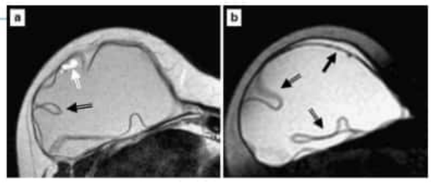


- MLO View: compress the breast along a plane of approximately 45 degrees extending from upper inner quadrant lower outer quadrant with the x-ray tube rotated parallel to the pectoralis muscle fibres. When done properly the MLO view shows both the axillary tail and the abdominal wall.
- CC View: The breast is directly kept on top of the xray cassette holder with the Xray tube positioned for superior to inferior imaging.
- Major role in Screening. All women over age of 40 yrs must undergo annual mammography for ruling out DCIS- MICROCALCIFICATION.
- BIRADS:



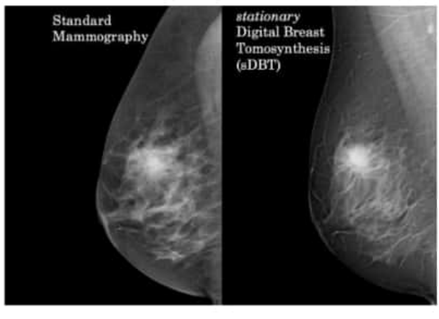
Final Assessment Categories			
Category		Management	Likelihood of cancer
0	Need additional imaging or prior examinations	Recall for additional imaging and/or await prior examinations	n/a
1	Negative	Routine screening	Essentially 0%
2	Benign	Routine screening	Essentially 0%
3	Probably Benign	Short interval follow-up (6 month) or continued	>0 % but ≤ 2%
4	Suspicious	Tissue diagnosis	4a. low suspicion for malignancy (>2% to ≤ 10%) 4b. moderate suspicion for malignancy (>10% to ≤ 50%) 4c. high suspicion for malignancy (>50% to <95%)
5	Highly suggestive of malignancy	Tissue diagnosis	≥95%
6	Known biopsy-proven	Surgical excision when clinical appropriate	n/a

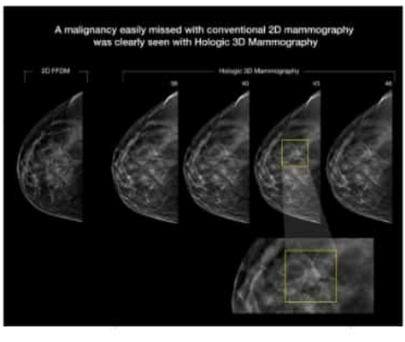
BREAST MRI



- Indicated in patients with breast implant
- Image shows an intracapsular rupture of a breast implant- **Linguine sign**

DIGITAL BREAT TOMOSYNTHESIS – DBT (3D Mammo)

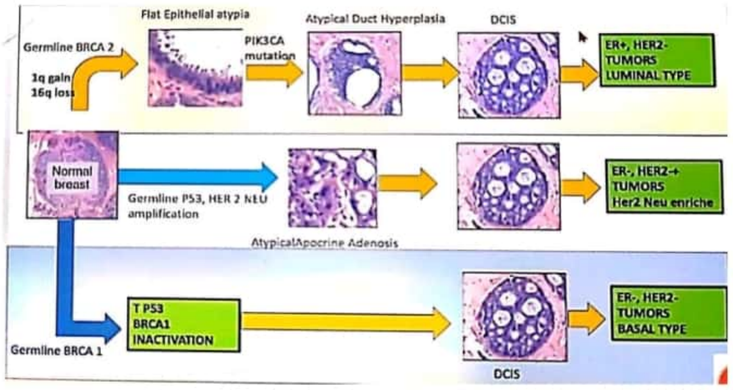


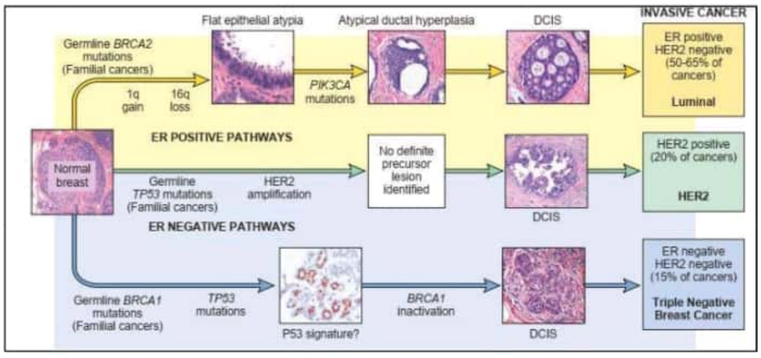


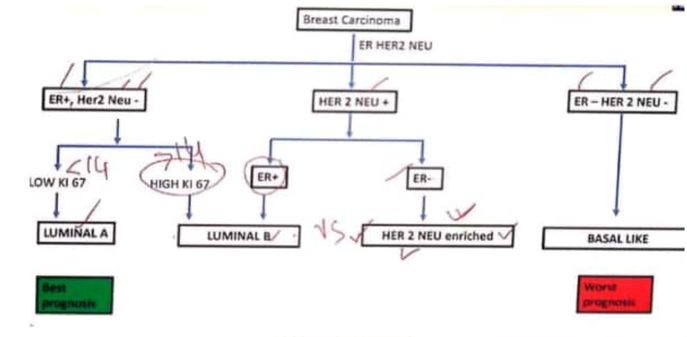
Ca Breast

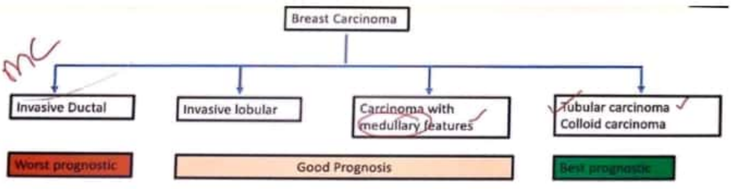
Pathogenesis

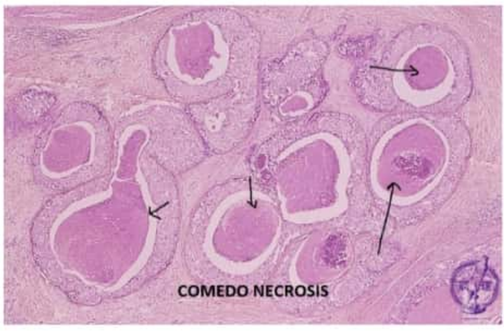
- **Pathological Dx: TRU CUT Biopsy is the gold standard investigation for Breast Pathologies.**

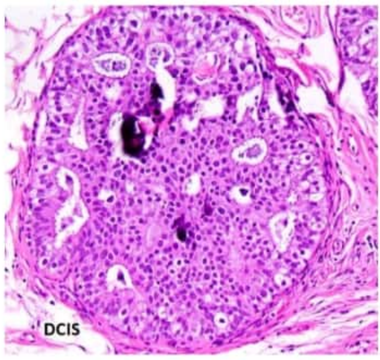




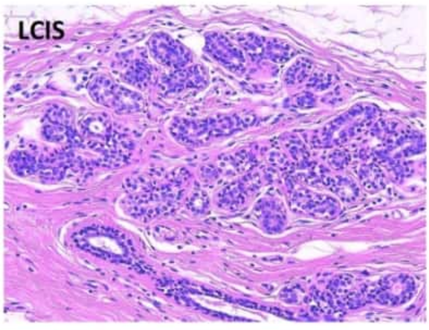


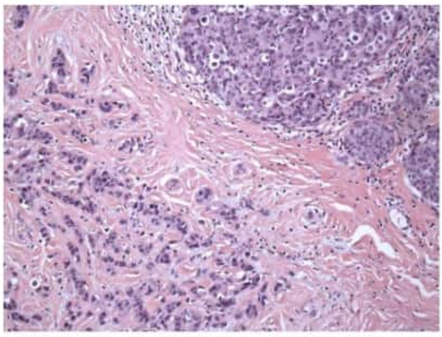






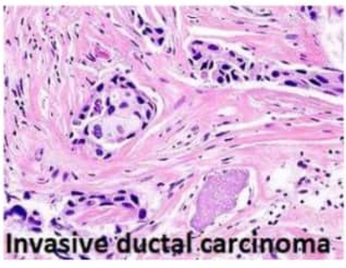
DCIS: Marker for invasion is p63



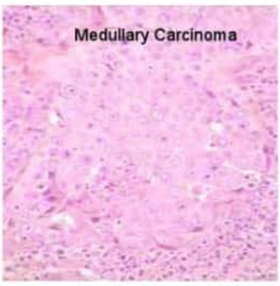


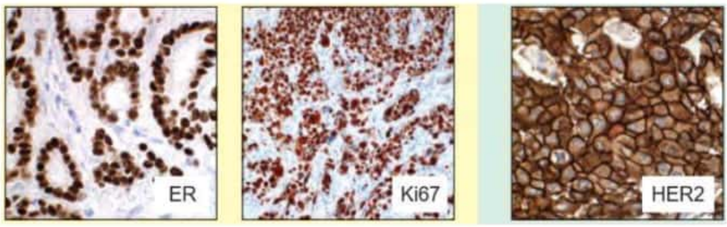
Invasive lobular carcinoma (left) adjacent to LCIS (right)

Invasive Lobular Ca- Indian File pattern



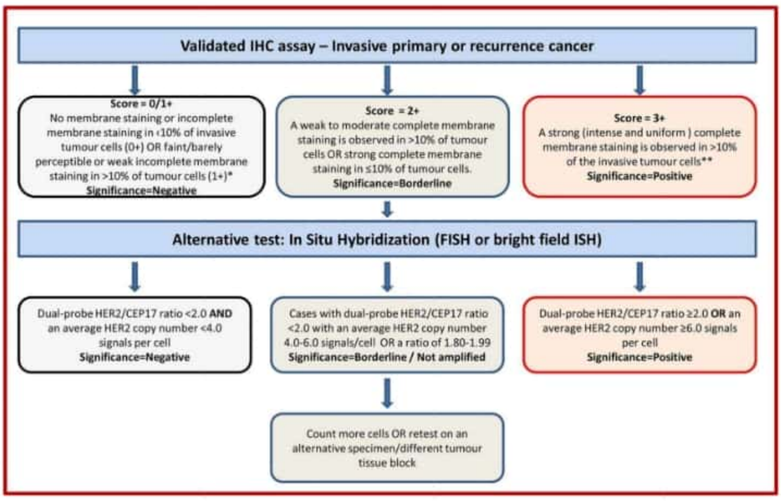
- Medullary Breast Carcinoma: Dense Lymphocytic infiltrates + Pleomorphic cells.



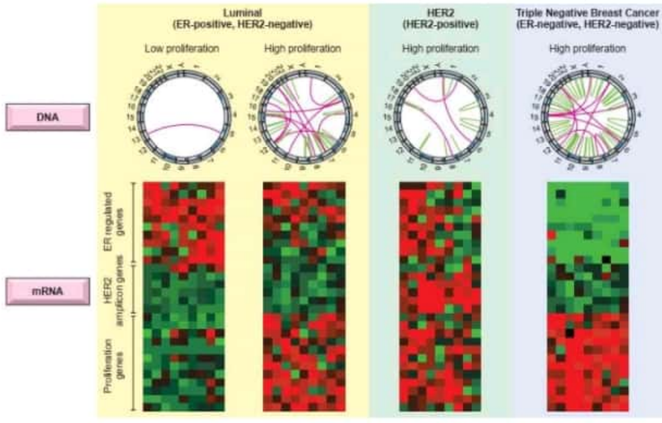


ER/ PR : Nuclear Pattern

HER2Neu: Membrane Pattern


Validated IHC assay – Invasive primary or recurrence cancer
Score = 0/1+
No membrane staining or incomplete membrane staining in <10% of invasive tumour cells (0+) OR faint/barely perceptible or weak incomplete membrane staining in >10% of tumour cells (1+)*
Significance=Negative
Score = 2+
A weak to moderate complete membrane staining is observed in >10% of tumour cells OR strong complete membrane staining in ≤10% of tumour cells.
Significance=Borderline
Score = 3+
A strong (intense and uniform) complete membrane staining is observed in >10% of the invasive tumour cells**
Significance=Positive
Alternative test: In Situ Hybridization (FISH or bright field ISH)
Dual-probe HER2/CEP17 ratio <2.0 AND an average HER2 copy number <4.0 signals per cell
Significance=Negative
Cases with dual-probe HER2/CEP17 ratio <2.0 with an average HER2 copy number 4.0-6.0 signals/cell OR a ratio of 1.80-1.99
Significance=Borderline / Not amplified
Dual-probe HER2/CEP17 ratio ≥2.0 OR an average HER2 copy number ≥6.0 signals per cell
Significance=Positive
Count more cells OR retest on an alternative specimen/different tumour tissue block

MICROARRAY FOR Breast Ca



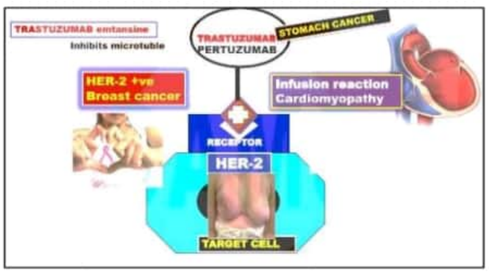
- Technique of evaluation of genes a/s cancer to evaluate the risk of recurrence
- Popularly used for Ca Breast, Ca Colon, Ca Stomach, Ca Esophagus
- Aim is to avoid chemotherapy
- Popular studies are: Oncotype DX, Mammaprint, Endopredict, GGI
- Inclusion Cretieria: Only for ER/PR+ and LN -ve status
- ONCOTYPE DX: 21 gene study
- Mammaprint: 70 gene study
- Endopredict: 12 gene study
- GGI: 97 Gene study
- PAM: 50 gene study

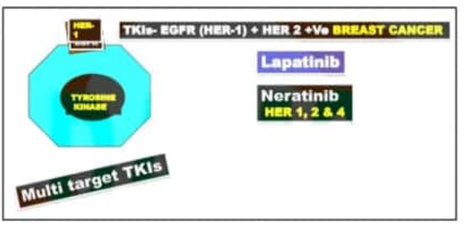
SURGERY:

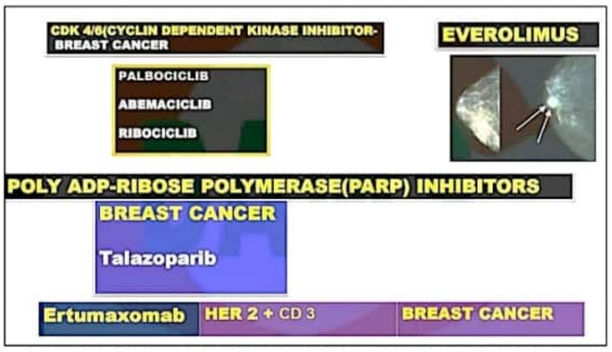
- Breast Conservation Surgery: Lumpectomy with an adequate 2mm tumour free margin
- Modified Radical Mastectomy: complete removal of breast and axially nodal clearance.

Post Surgical Management

- Chemotherapy; required for tumors >1cm in size or >5mm (with LN+ve or high risk groups)
  Most popular regimens include 4 cycles of ADRIAMYCIN and 4 cycles of PLATINUM or TAXANES
- Radiotherapy: For all LABC tumors and few cases of EBC, 50 Gy at weekly divided doses in 6 weeks.
- APBI: Accelerated partial breast irradiation is a low dose brachytherapy for post BCS status patients with: Age 50-60 yrs, T size <2cm; LN: adequate tumor free margins, ductal ca on pathology.







ANTI -CANCER DRUGS OVERALL

BLOOD MALIGNANCIES

ALL

Induction combination chemotherapy:
Vincristine,
Prednisone, daunorubicin,
asparaginase,
Intrathecal Methotrexate

Maintenance chemotherapy:
Methotrexate,
6-mercaptopurine

Philadelphia chromosome–positive ALL:
Add imatinib or dasatinib or nilotinib to above regimen

AML

Combination chemotherapy:

Cytarabine, daunorubicin or

Cytarabine, idarubicin or

Cytarabine, daunomycin, cladribine

AML

ENASIDENIB, IVOSIDENIB

Isocitrate dehydrogenase2 (IDH2) inhibitor

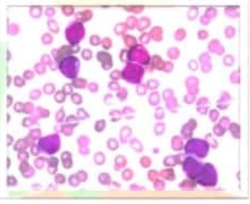
GLASDEGIB

HEDGEHOG SIGNALING PATHWAY INHIBITOR

GEMTUZUMAB Ozogamicin- CD 33

MIDOSTAURIN

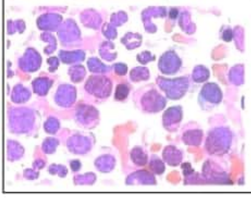
Multi kinase inhibitor



Gilteritinib

CML

Hydroxyurea
INF- α
BUSULFAN



PDGFR

TYROSINE KINASE

BCR-ABL TKIs- CML

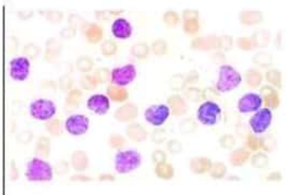
IMATINIB
CML (DOC)
I generation

DASATINIB
NILOTINIB
BOSUTINIB
2nd-generation inhibitors

Radotinib
Ponatinib
Bafetinib
3rd-generation inhibitors

OMACETAXINE

CLL



CHLORAMBUCIL
CYCLOPHOSPHAMIDE
FLUDARABINE – Anti metabolite

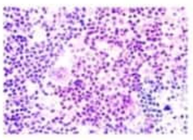
CD-20
RITUXIMAB-
OFATUMUMAB
OBINUTUZUMAB

CD-52
ALEMTUZUMAB-

BRUTON TKI- IBRUTINIB

VENETOCLAX
B- CELL LYMPHOMA-2 (BCL-2) PROTEIN INHIBITOR

HODGKIN'S LYMPHOMA



MOPP
Mechlorethamine
onchovin(vincristine)
procarbazine
prednisone

ABVD
Doxorubicin
Bleomycin
Vinblastine
Decarbazine

STANFORD V

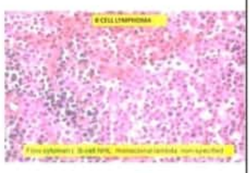
Doxorubicin, vinblastine, mechlorethamine, etoposide, vincristine, bleomycin, prednisone

Brentuximab vedotin- CD30
Nivolumab, Pembrolizumab

Bleomycin, etoposide, doxorubicin, cyclophosphamide, vincristine, procarbazine, prednisone (BEACOPP)

NON HODGKIN'S LYMPHOMA

CHOP
Cyclophosphamide
Doxorubicin
Vincristine
Prednisone

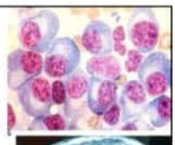


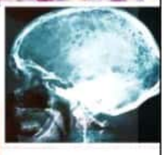
CHOP-R
CHOP + RITUXIMAB

NON HODGKIN'S LYMPHOMA (LOW GRADE)-
Cyclophosphamide, vincristine, prednisone, rituximab (CVP-R)

Multiple myeloma

Melphalon
Thalidomide, Lenolidomide,
Pomalidomide
Cyclophosphamide
Dexamethasone
Bortezomib
Carfilzomib
Ixazomib
PROTEOSOME INHIBITOR





Panobinostat
Histone De Acetylases (HDA) enzyme inhibitor

Elotuzumab- CD 319,
Daratumumab- CD 38

Stem cell transplantation

Polycythemia vera

Phlebotomy, hydroxyurea, aspirin

Ruxolitinib
Radiophosphorus 32p
Interferon-alpha

SYSTEMIC NEOPLASMS

BRAIN TUMOR

Nitrosoureas
Temozolamide
Procarbazine
Bevacizumab

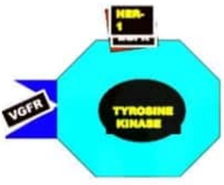
HEAD & NECK CANCER

Combination chemotherapy:
Cisplatin, fluorouracil, or
Paclitaxel, carboplatin, or
Docetaxel, cisplatin, fluorouracil, or
Cisplatin or cetuximab with radiation therapy

Pembrolizumab, nivolumab
Afatinib

Duligotuzumab — HER 1 + HER 3

THYROID CANCER



Radioiodine I 131
Lenvatinib
Sorafenib

Vandetanib
Cabozantinib

Medullary cancer of thyroid

Esophageal and esophagogastric junction cancer

Combination chemotherapy:
Cisplatin, fluorouracil, or
Paclitaxel, carboplatin, or
Oxaliplatin, fluorouracil,

Trastuzumab,

Ramucirumab (esophagogastric junction cancer),

Pembrolizumab

TKIs- GIST

IMATINIB- C KIT

SUNITINIB

REGORAFENIB

AVAPRITINIB

EPIRUBICIN,
CISPLATIN,
FLUOROURACIL,

Gastric cancer

Ramucirumab

PEMBROLIZUMAB

ERLOTINIB
SUNITINIB

Combination chemotherapy:

Gemcitabine, nab-paclitaxel, or

5-Fluorouracil, leucovorin, irinotecan, oxaliplatin (FOLFIRINOX)

Single-agent chemotherapy: GEMCITABINE

Adenocarcinoma-pancreas

Hepatocellular cancer

Single-agent chemotherapy:

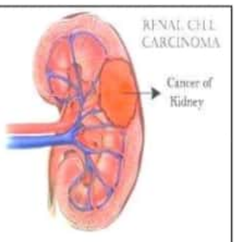
Sorafenib or lenvatinib

Regorafenib, nivolumab, ramucirumab, pembrolizumab

TYROSINE KINASE INHIBITORS

VGFR BLOCKER-
Sorafinib
Sunitinib

VGFR+ PDGFR BLOCKER
Axitinib
Pazopanib



VGFR BLOCKING MAB
Bevacizumab

COLO RECTAL CANCER

Combination chemotherapy:

Fluorouracil, leucovorin, oxaliplatin (FOLFOX),
or

Capecitabine, oxaliplatin (CapeOx),
Or

Fluorouracil, leucovorin, irinotecan (FOLFIRI)

each regimen with or without BEVACIZUMAB

Catumaxomab	Epithelial Cell Adhension Molecule(EpCAM) + CD 3	Malignant ascities

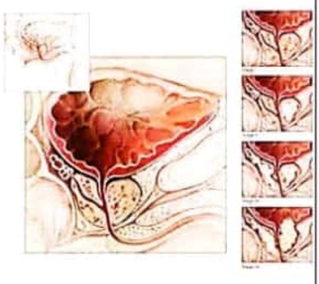
PROSTATE CANCER

LHRH ANALOGUE(LEUPROLIDE) + ANTI ANDROGEN (FLUTAMIDE..)

DOCETAXAL +
PREDNISOLONE
MITOXANTRONE

sipuleucel-T (cell-based cancer immunotherapy

Radium (Ra)-223



TESTICULAR CANCER

Combination chemotherapy:

Cisplatin, etoposide (EP), or

Bleomycin, etoposide, cisplatin (BEP), or

Etoposide, mesna, ifosfamide, cisplatin

UROTHELIAL CARCINOMA

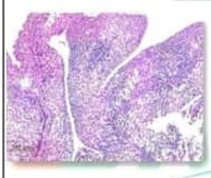
Atezolizumab

NSCL Cancer

Durvalumab

mune check point inhibitor, targeting Against programmed death ligand-1 (PDL1)

DURVALUMAB



TARGET- PROGRAMMED DEATH LIGAND

urothelial bladder cancer

Atezolizumab –urothelial carcinoma

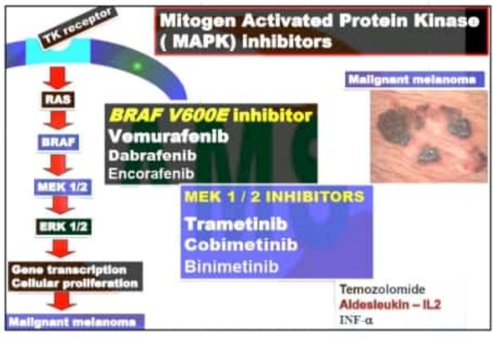
Avelumab – Merkel cell carcinoma

OVARIAN CANCER

Combination chemotherapy:

Paclitaxel, carboplatin with or without bevacizumab,
or
Docetaxel, carboplatin,
or
Paclitaxel, cisplatin

Poly ADP ribose polymerase (PARP) inhibirors
Olaparib
Rucaparib
Niraparib



MALIGNANT MELANOMA

IPILIMUMAB	Cytotoxic T Lymphocyte–Associated Protein 4 (CTLA- 4)
NIVOLUMAB PEMBROLIZUMAB	immune check point inhibitor, Programmed Death-1 (PD-1)

Mycosis Fungoides; Sézary Syndrome

MOGAMULIZUMAB

chemokine receptor 4 (CCR4) inhibitor

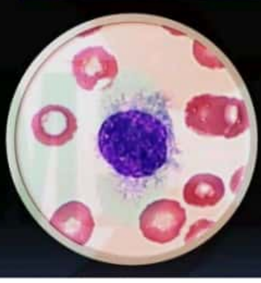
Denilukin diftitox

Histone De Acetylase inhibitor

VORINOSTAT
ROMIDEPSIN

HAIRY CELL LEUKEMIA

MOXETUMOMAB
CLADRIBINE
PENTOSTATIN



OSTEOSARCOMA

Cisplatin, doxorubicin,

or

Methotrexate, doxorubicin, cisplatin (MAP)

WILMS TUMOR

VINCRITSTINE + DACTINOMYCIN + DOXORUBICIN

Kaposi sarcoma

Liposomal doxorubicin

or

liposomal daunorubicin

or

paclitaxel

VINCRISTINE

MECHLORETHAMINE

DOXORUBICIN

BLEOMYCIN

Muco cutaneous reaction (flagellated pigmentation on skin)

Cerebellar ataxia

Cytarabine (ARA-C) CYTOSINE ARABINOSIDE

PULMONARY FIBROSIS

BLEOMYCIN BUSULFAN

HAND FOOT SYNDROME

Capecitabine 5-Flurouracil Doxorubicin Interleukin-2 Pemetrexed

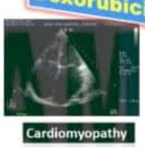
CYCLO PHOSPHAMIDE

Haemorrhagic cystitis

Mesna

ANTIDOTE

Doxorubicin



Cardiomyopathy

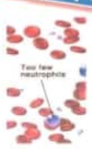
DEXRAZOXANE

Cisplatin

Nephrotoxic

Amifostine

Neutropenia



CSF

ANTIDOTE

Anemia

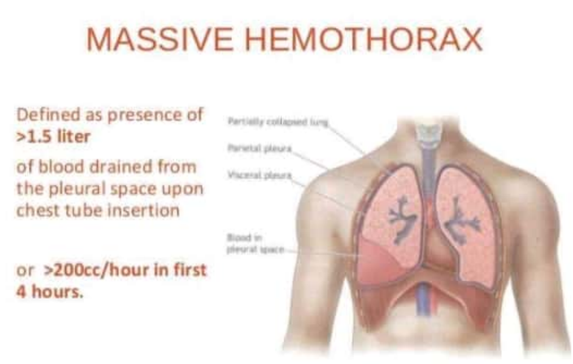
Epoietin

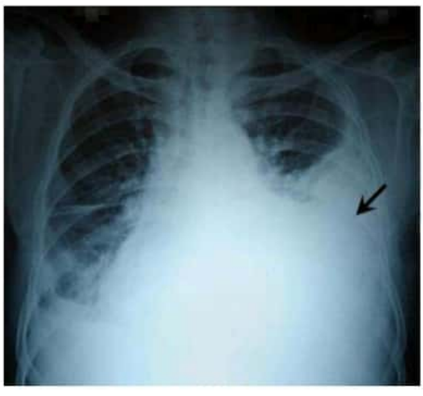
Thrombo cytopenia

Oprelvekin

Hemothorax (Chest Trauma)

Case: A 24-year-old woman is a passenger in a motor vehicle collision. On examination the left hemithorax is dull on percussion and breath sounds are absent on that side. There is tenderness over the 3rd and 4th rib. What is the most probable diagnosis?

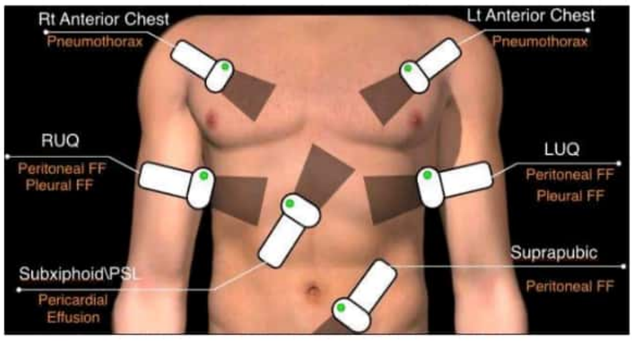

MASSIVE HEMOTHORAX
Defined as presence of >1.5 liter of blood drained from the pleural space upon chest tube insertion
or >200cc/hour in first 4 hours.
Partially collapsed lung
Parietal pleura
Visceral pleura
Blood in pleural space



Xray Findings:

- CP angle is blunted
- Diaphragm is obscured
- Convex shadow on the fluid border
- H/o Trauma indicates it is blood
- 100-200ml fluid is required in pleura to appear on CXR

Diagnosis: Clinically/CXR/**eFAST**



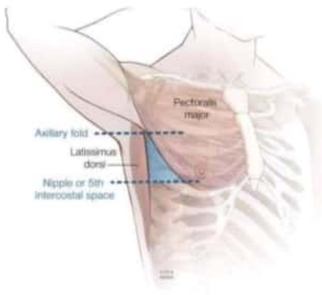
Management: Drain blood by **Intercostal Chest Tube Drain (ICD)**

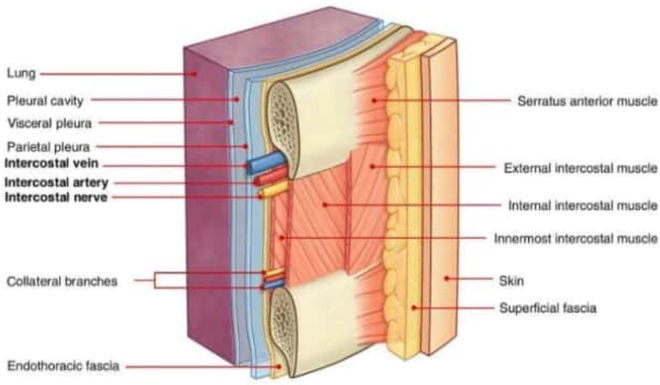
- Site: 5th I/C Space, just anterior to mid-axillary line, just above the upper border of the lower rib
- Size of ICD for Hemothorax: 28-32 Fr
- Indications of removal: a) Stable b) Inflated lung on CXR c) <200ml/24 hr output

Triangle of Safety

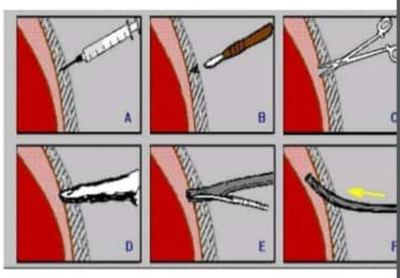
Boundaries: -

- Anterior – lateral border of pectoralis major
- Posterior – lateral border of Lat dorsi
- Inferior – horizontal line drawn from 5th IC space.





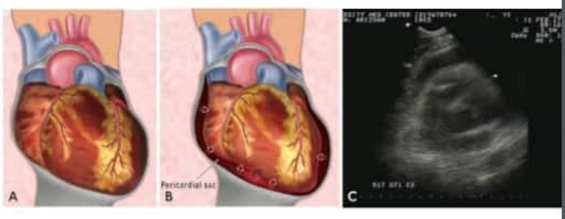
Insertion of chest tube



Indications of Thoracotomy:

- >1500ml blood in the ICD
- >200ml/hr blood for 2-4 hours.
- Hemodynamically unstable
- Associated Cardiac/Vascular injuries

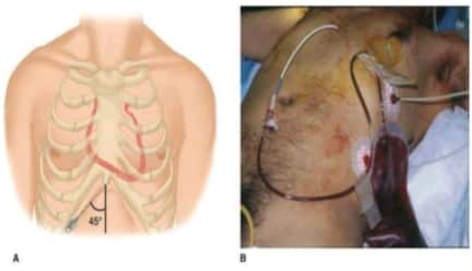
CARDIAC TEMPONADE



- Accumulation of fluid in pericardium
- 50-60 ml blood can also cause it
- Pathology: Accumulation of fluid compresses the SVC and IVC and the patient goes in Obstruction Shock
- Dx: **eFAST**
  - 1st keep the probe in sub-xiphoid area then tilt it towards the xiphoid process sub-costally.
- C/F: Muffled Heart Sounds, Distended Neck veins, Hypotension **[Beck's triad]**
  Tachycardia also (Not a part of the triad)

NOTES NEXT DOOR

- Treatment: PERICARDIOCENTESIS
  - Needle insertion in the subxiphoid area, 45^{O} to the vertical, 5^{O} to the skin, towards the left shoulder
- Definitive Sx: Thoracotomy + Pericardial Repair

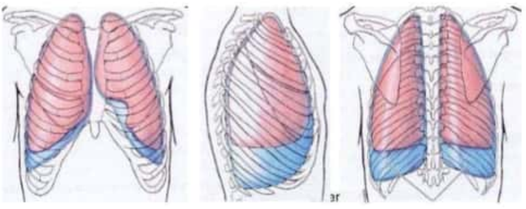


Pneumothorax

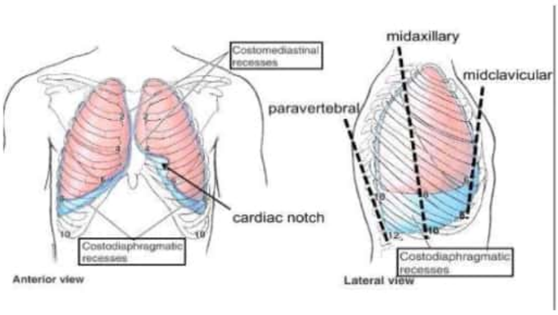
A 25-year-old male sustained injury in a motor vehicle collision and presented to the emergency with shortness of breath and chest pain. His pulse rate was 120 bpm and blood pressure was 80/50 mmHg. His respiratory rate was 28/min. On examination the right hemithorax is hyper-resonant on percussion and breath sounds are absent on the on the right side. The most probable diagnosis is?

Pleural Anatomy

- Visceral & Parietal Pleura
- Parietal pleura can further be divided into:
  - Diaphragmatic Pleura
  - Costal Pleura
  - Mediastinal Pleura
  - Cervical Pleura
- Two recesses:
  - Costo-mediastinal recess
  - Costo-diaphragmatic recess



	Midclavicular	Midaxillary	Paravertebral
Lung	6th rib	8th rib	10th rib
Pleura	8th rib	10th rib	12th rib

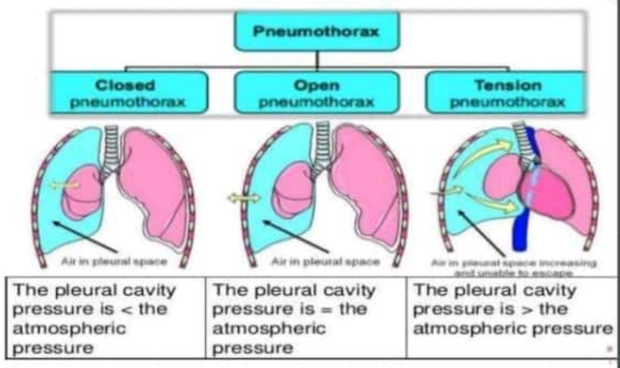


Types of Pneumothorax

1. **Simple/Closed:** Air from the lungs escapes into the pleura. No defect in external chest wall.

2. **Open**: Defect is present in the external chest wall, with size >2/3rd of the diameter of trachea (~2.5 cm). Loss of negative intrapleural pressure

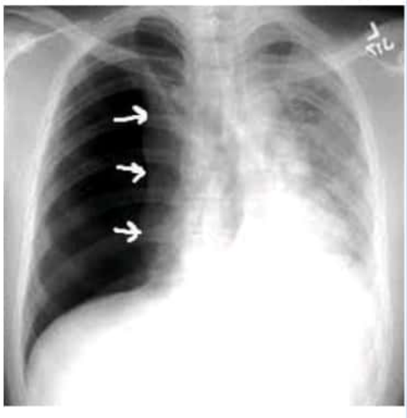
3. **Tension:** One-way defect in the chest wall. Causes Mediastinal shift and Heart rotation causing compression of SVC & IVC leading to **OBSTRUCTIVE SHOCK** and will present with **hypotension.**

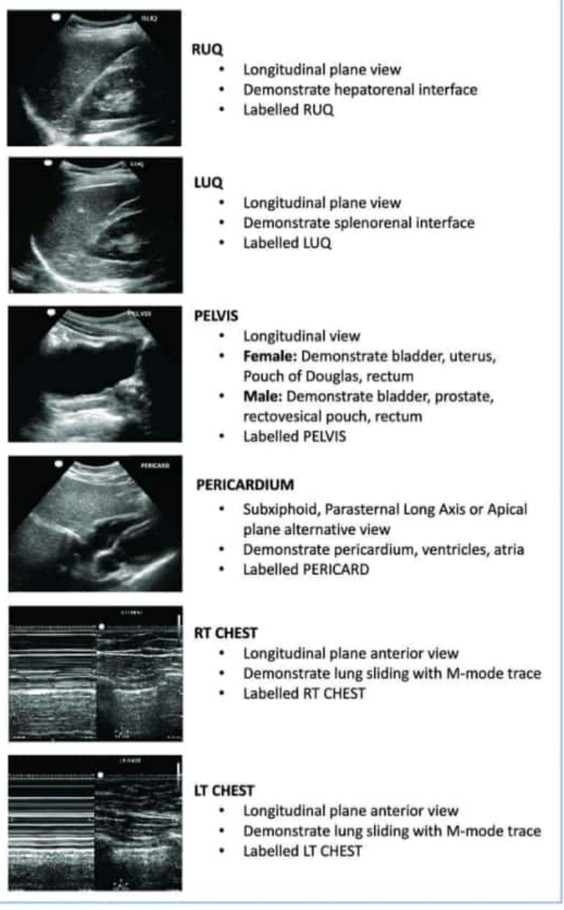


Diagnosis: **eFAST** **{USG}** (CXR not recommended because it's an emergency situation)

Chest Xray Findings

- Hyper-lucent area
- No broncho-vascular markings
- Collapsed lung can be seen
- Mediastinum and trachea pushed to C/L sides
- Most Imp Sign: **Visceral Pleural Line Sign**
  (WHITE ARROWS) Shows that air is in between visceral and parietal pleura. Helps to differentiate pneumothorax from emphysematous bullae.





RUQ
- Longitudinal plane view
- Demonstrate hepatorenal interface
- Labelled RUQ

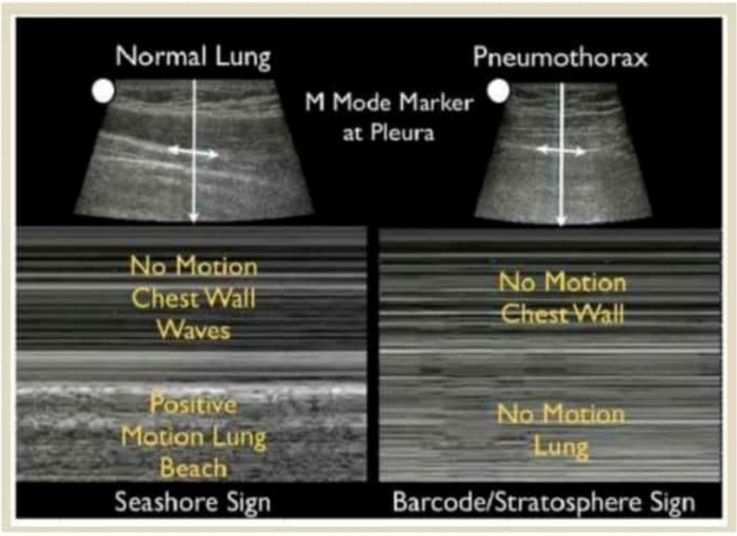
LUQ
- Longitudinal plane view
- Demonstrate splenorenal interface
- Labelled LUQ

PELVIS
- Longitudinal view
- **Female:** Demonstrate bladder, uterus, Pouch of Douglas, rectum
- **Male:** Demonstrate bladder, prostate, rectovesical pouch, rectum
- Labelled PELVIS

PERICARDIUM
- Subxiphoid, Parasternal Long Axis or Apical plane alternative view
- Demonstrate pericardium, ventricles, atria
- Labelled PERICARD

RT CHEST
- Longitudinal plane anterior view
- Demonstrate lung sliding with M-mode trace
- Labelled RT CHEST

LT CHEST
- Longitudinal plane anterior view
- Demonstrate lung sliding with M-mode trace
- Labelled LT CHEST



Treatment:

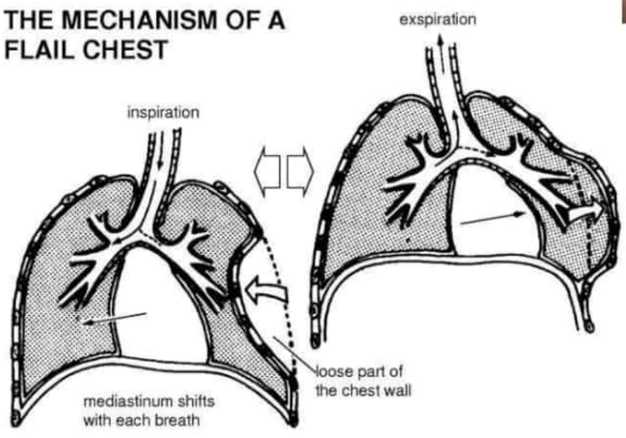
- Closed Pneumothorax
  - Conservative Management
  - ICD Insertion
- Open Pneumothorax
  - 3-sided Occlusive Dressing
    (Kept open to make it a one-way valve from where only the air comes out as it closes during inspiration)
  - ICD Insertion
- Tension Pneumothorax:
  - 1st: Needle Thoracocentesis
    - Children: 2nd ICS Mid- Clavicular Line
    - Adults: 5th ICS Mid- Clavicular Line
    - Needle: 14 G
  - ICD insertion
    - Size: 24 -28 Fr

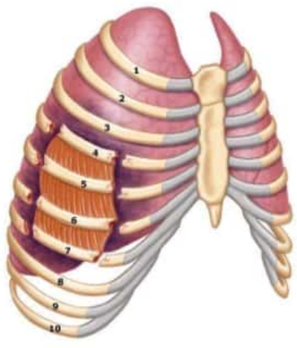
FLIAL CHEST

# of 2 or more consecutive ribs and at least 2 fractures on each rib.

C/F- PARADOXICAL MOVEMENT

The inward movement of the fragment can cause **lung contusion**.





Treatment: Nothing to be done for fracture

- If no respiratory distress: Oxygen by mask + Epidural Analgesics
- If patient in respiratory distress: Intubate and give IPPV.

DEADLY DOZEN OF CHEST TRAUMA	
IMMEDIATELY LIFE THREATENING	**POTENTIALLY LIFE THREATENING**
1. Airway Obstruction	1. Flail Chest
2. Tension Pneumothorax	2. Aortic Injuries
3. Open Pneumothorax	3. Myocardial Infarction
4. Massive Hemothorax	4. Diaphragmatic injuries
5. Tracheobronchial Injuries	5. Esophageal injuries
6. Cardiac Tamponade	6. Pulmonary Contusions
Should be handled in PRIMARY survey	Should be handled in SECONDARY survey

RUPTURED ABDOMINAL AORTIC ANEURYSM

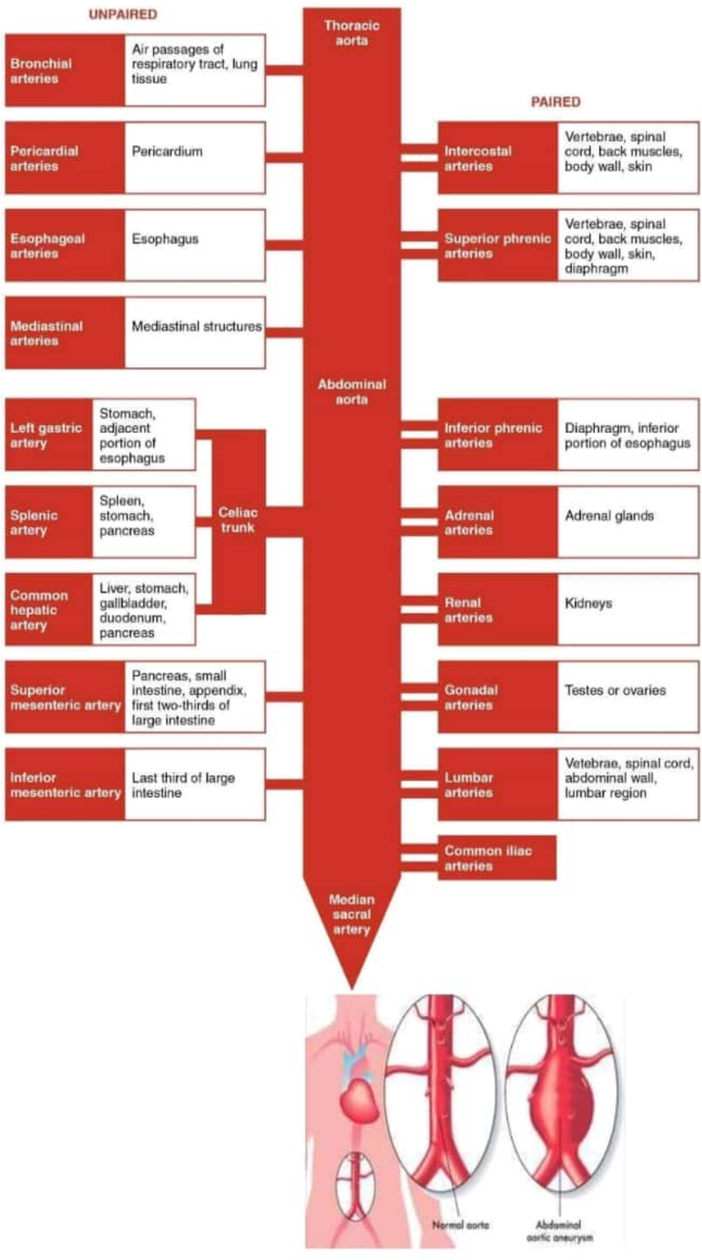
Case: A 70-year-old male with atherosclerosis and abdominal lump complains of back and abdominal pain and then collapses. His blood pressure is 90/40 mmHg. The patient denies a history of peptic ulcer or alcohol abuse. What is the most likely diagnosis?

ANATOMY

Branches of Abdominal Aorta:

- Celiac Trunk at T12
- SMA at L1
- IMA at L3 level

- Abdominal aorta (Lumbar artery) supplies only 4 L1 to L4 vertebrae before bifurcation at L4.



- **True Aneurysm**

Abnormal dilation of artery due to weakened vessel wall

- **False Aneurysm**

Blood vessels is injured & blood leaks into surrounding connective tissue.

→ M/C site of AAA is – **Infrarenal Abdominal Aorta**

→ M/C Cause: Atherosclerosis

→ Screening for AAA is done in western countries because of its deadliest complications: Rupture of the AAA (M/C- Posterior Rupture)

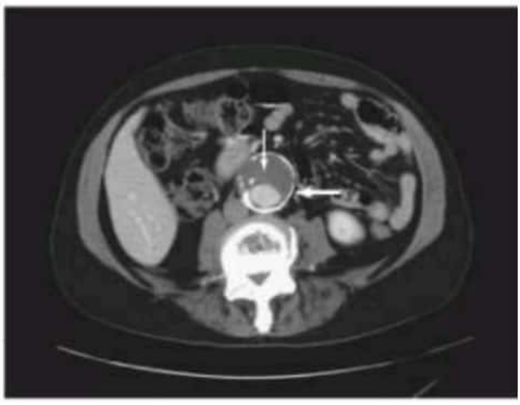
- Posterior Rupture- 80%
- Anterior Rupture- 20% --- Mostly fatal

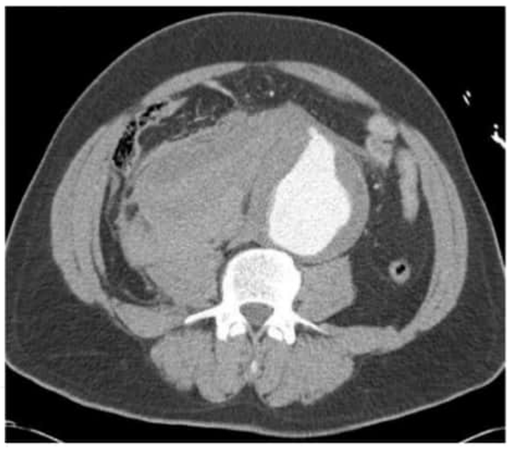
Other Complications:

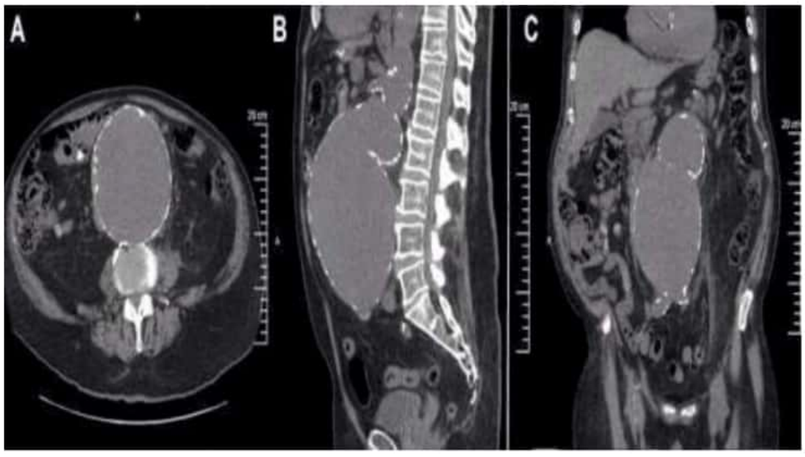
- Aorto-caval fistula → High Output Cardiac Failure
- Aorto-enteric fistula → Fistula with 3rd/4th part of Duodenum → Severe Lower GI Bleed

Investigations

- **Screening:** USG
- **IOC:** CT Angiogram
  - Dilatation of the aorta
  - Hematoma involving the psoas muscle
  - Aorta starts to drape over the vertebra **(k/a DRAPING SIGN)**
  - Aortic Wall calcification



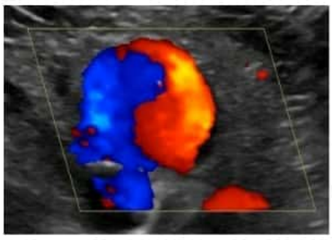




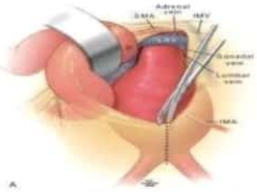
Benefits of CT-Angiogram over a Catheter Angiogram

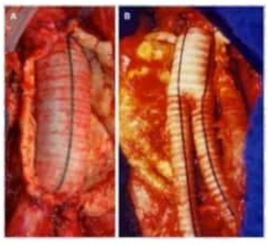
- Uses an IV dye and is NON-invasive
- Estimate both thrombosed and non-thrombosed part

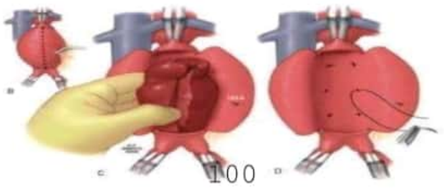
Catheter-Angiogram



Psuedo-Aneurysm shows to and fro movement on a doppler k/a **YIN YANG SIGN**

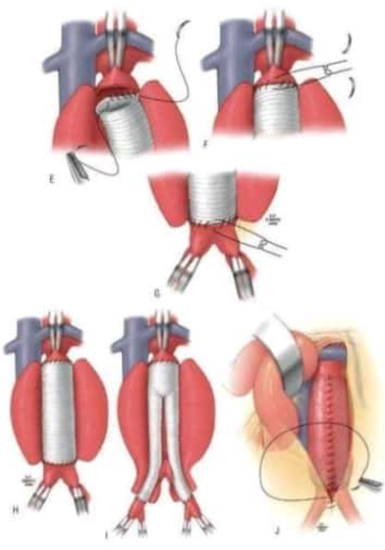






NOTES NEXT DOOR

Estimated Annual Rupture Risk	
AAA DIAMETER (cm)	RUPTURE RISK (%/yr)
<4	0
4-5	0.5-5
5-6	3-15
6-7	10-20
7-8	20-40
>8	30-50



Treatment: Surgery

Indications of surgery:

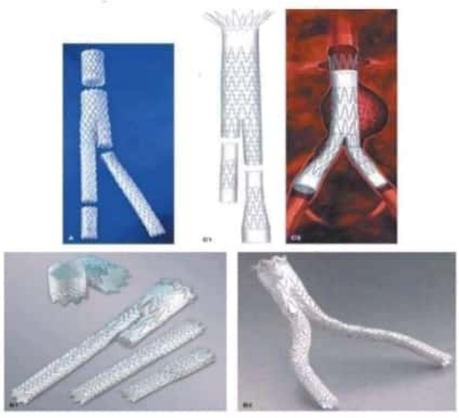
- Compression Symptoms
- Distal Emboli
- **Asymptomatic patients only when diameter >5.5 cms**
  or rate of growth is >0.5cm/6month or >1cm/year

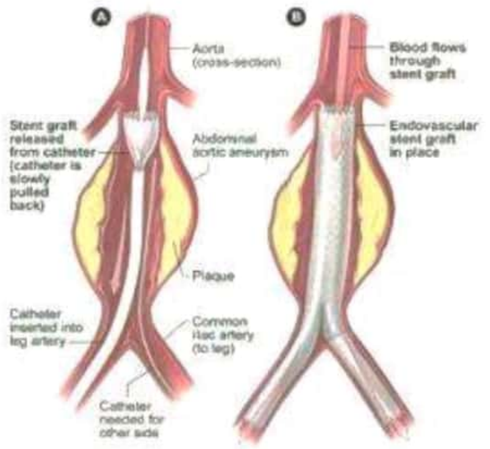
Dacron Graft is used commonly

New: Endovascular Aneurysm Repair (EVAR)

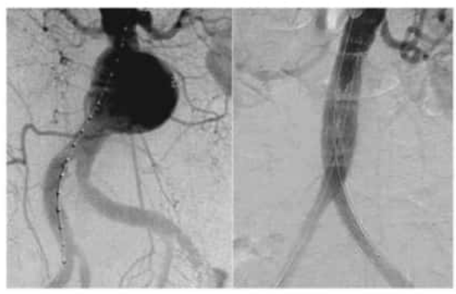
Stent is made up of:

- Nitinol
- Polyester

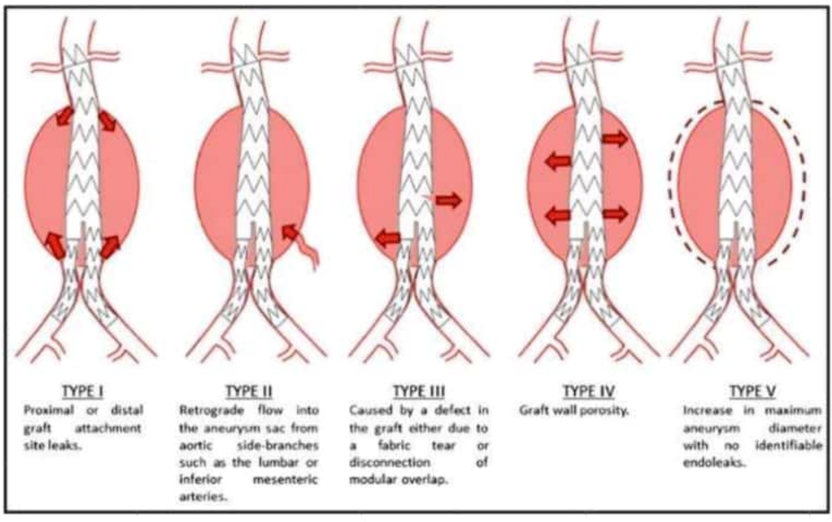




Before & After EVAR →→→



Complications of EVAR: **Endoleak** (Type 5 is aka ENDOTENSION)

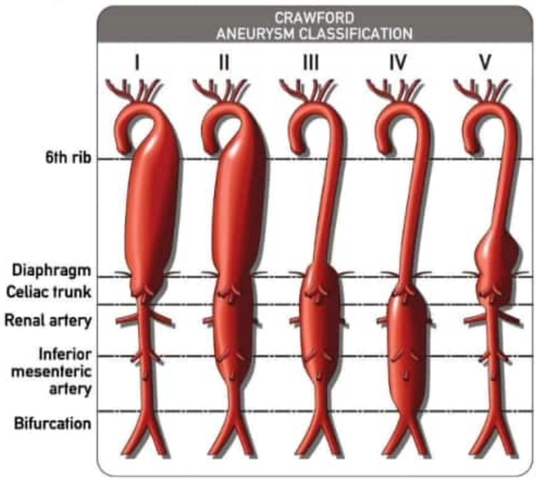

TYPE I
Proximal or distal graft attachment site leaks.
TYPE II
Retrograde flow into the aneurysm sac from aortic side-branches such as the lumbar or inferior mesenteric arteries.
TYPE III
Caused by a defect in the graft either due to a fabric tear or disconnection of modular overlap.
TYPE IV
Graft wall porosity.
TYPE V
Increase in maximum aneurysm diameter with no identifiable endoleaks.

Mx: Type 1 & 3 – Urgent Intervention (Change graft)
Type 2, 4, 5 – Conservative management

Thoracic Artery Aneurysm

TAAA Classification (Crawford classification , 1986)

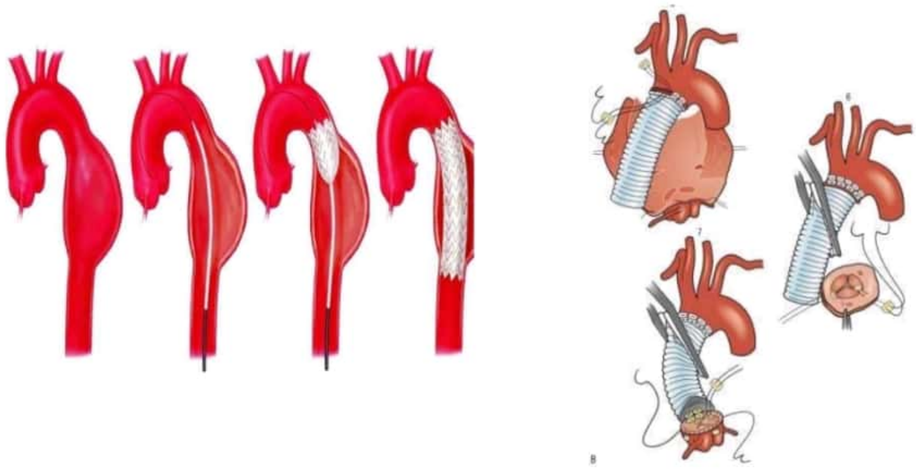
I. Below L SCA to above renal arteries.
II. Below L SCA and including infrarenal aorta to the bifurcation.
III. 6th intercostal space to bifurcation.
IV. 12th intercostal space to bifurcation.
V. 6th intercostal space to above renal arteries.



Q. MOST Extensive type is *TYPE 2*

Treatment: Open or Endovascular Repair

TEVAR: Thoracic EVAR



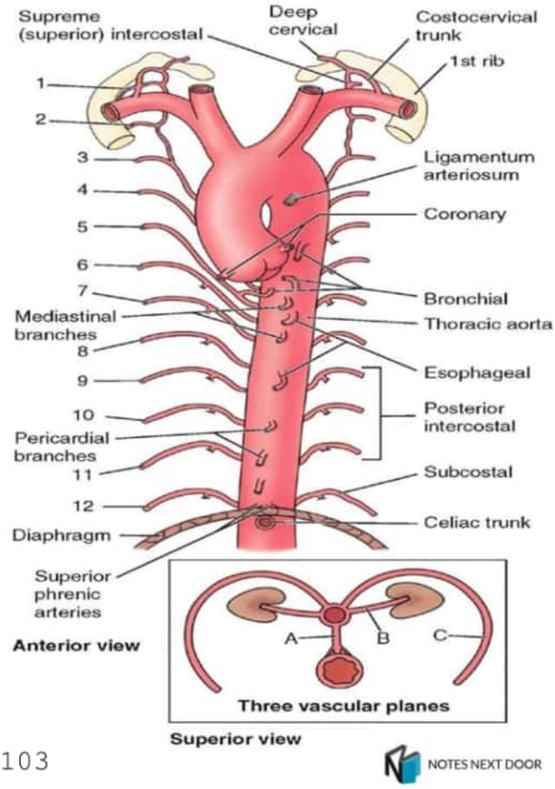
AORTIC DISSECTION

Case: A 45-year-old man with poorly controlled hypertension presents with severe chest pain radiating to his back. An ECG demonstrates no significant abnormalities. Serum amylase is elevated and serum lipase is within normal levels. What is the most probable diagnosis?

ANATOMY

Branches of Desc thoracic aorta

- Pericardial
- Mediastinal
- Esophageal
- Bronchial
- 9 Posterior IC arteries
- Rt & Lt Superior phrenic artery
- Rt & Lt Subcostal artery



Histology

- *Tunica Intima*
  - Endothelium (Simple squamous epithelium)
  - Subendothelial Connective tissue
  - Internal Elastic Lamina
- *Tunica Media*
  - In aorta contains large number of elastic fibres
  - In muscular artery contains smooth muscles

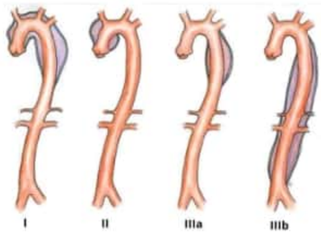
NOTES NEXT DOOR

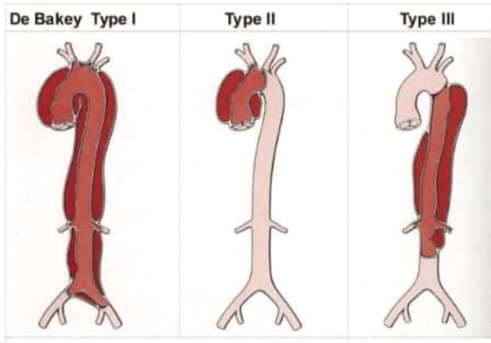
- *Tunica Adventitia*
  - Contains nerves and vessels (Vasa vasorum + Nervi vasorum)

Causes of Aortic Dissection

- Hypertension- M/C
- Cystic Medial Necrosis
- Connective Tissue D/o
  - Ehler-Danlos Syndrome
  - Marfan's Syndrome

Standford Classification





De Bakey

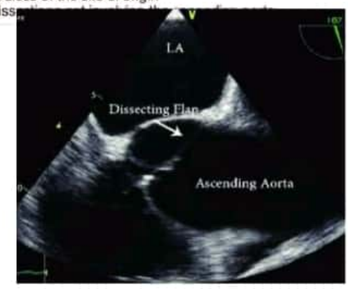
Type I	Originates in the ascending aorta, propagates at least to the aortic arch and often beyond it distally
Type II	Originates in and is confined to the ascending aorta
Type III	Originates in the descending aorta and extends distally down the aorta or, rarely, retrograde into the aortic arch and ascending aorta

Stanford

Type A	All dissections involving the ascending aorta, regardless of the site of origin
Type B	All diss…

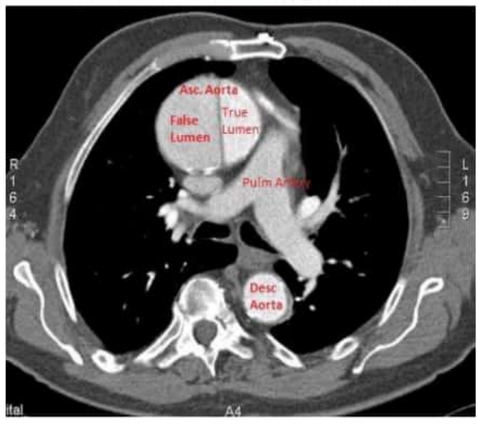
Radiology

- Unstable Patient: Trans-Esophageal Echocardiography
- Stable Patient: CT- Angiogram



CT Angiogram Findings:

- False Lumen: Outer, Larger, Less Enhancing because of less dye going into it
- **COBWEB sign:** strands of disrupted media fibres in the false lumen

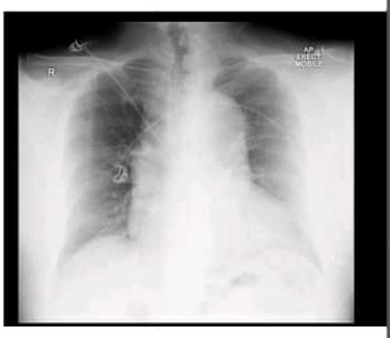


→Standford Type A Aortic Dissection is the real emergency situation

Descending Aorta Dissection
(Standford Type B)
Medical Management can be tried

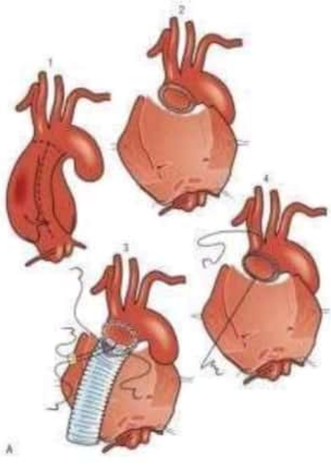
Xray has no diagnostic role but findings which can be seen in an CXR are:

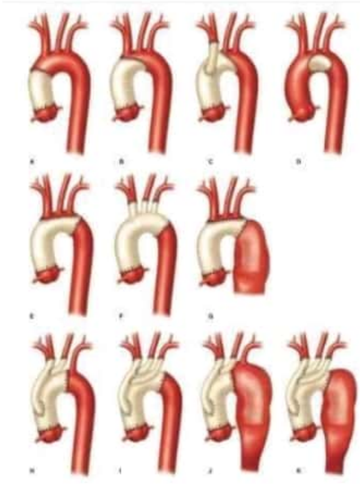
- Mediastinal widening
- Intimal Calcification sometime
- Progressive aortic enlargement



Treatment

- Initial Mx (Next Step): BETA BLOCKERS to stabilize the patient and relieve the tension from aortic wall.
- In Standford Type A- Surgery: Repair
- In Standford Type B- Antihypertensive medical mx.



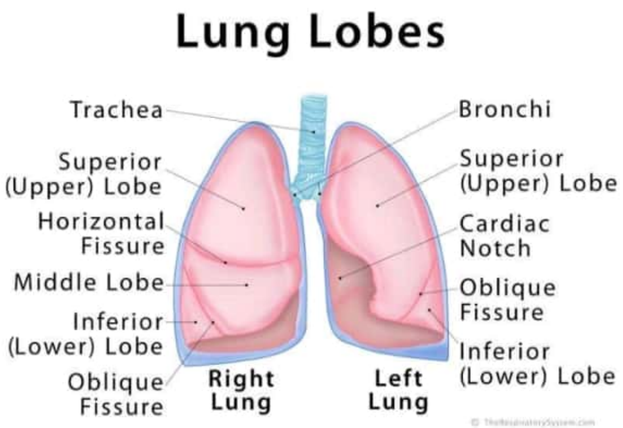


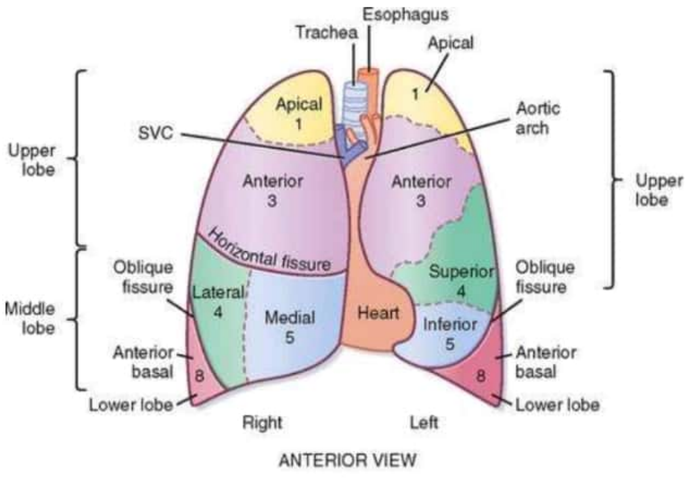
LUNG CANCER

Case: A 45-year-old man is admitted to the hospital with severe dyspnea with a 40 pack-year history of smoking. Physical examination reveals that the patient has miosis of the pupil, partial ptosis of the eyelid, and anhydrosis of the face. Most likely diagnosis?

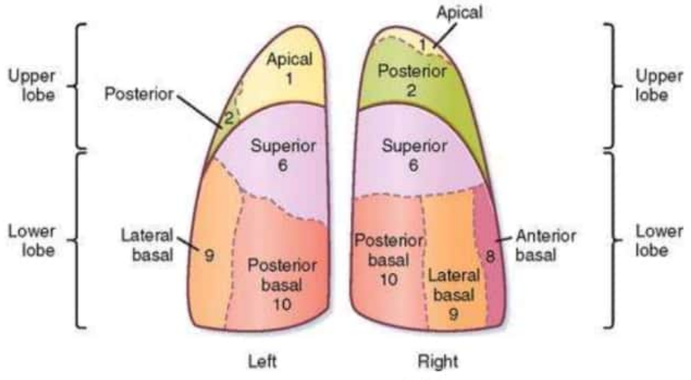
ANATOMY

Bronchopulmonary segment:
Unit of the lung which is supplied by one TERTIARY BRONCHUS





ANTERIOR VIEW



- Involvement of lower trunk of Brachial plexus
- Involvement of sympathetic as they emerge from T1 to reach the superior cervical ganglion
- **Horner's syndrome** – miosis, ptosis, anhydrosis, enophthalmos.

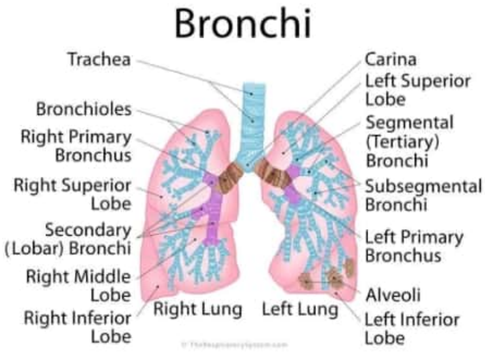


TABLE 55.6 The international tumour–node–metastasis (TNM) staging system.

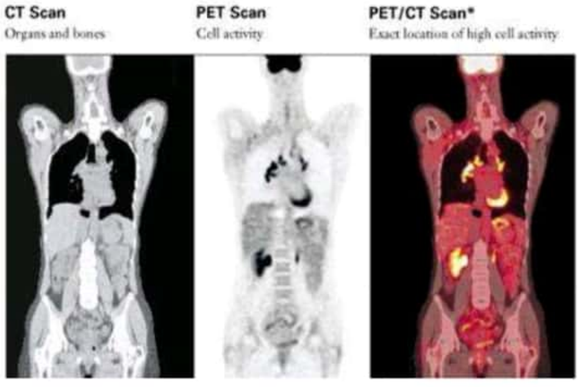
T: Primary tumour	
Tx	Primary tumour cannot be assessed or tumour proven by presence of malignant cells in sputum or bronchial washings but not visualised by imaging or bronchoscopy
T0	No evidence of primary tumour
Tis	Carcinoma *in situ*
T1	Tumour ≤3 cm in greatest dimension surrounded by lung or visceral pleura without bronchoscopic evidence of invasion more proximal than the lobar bronchus (i.e. not in the main bronchus)[a]
T1a(mi)	Minimally invasive adenocarcinoma[b]
T1a	Tumour 1 cm in greatest dimension[a]
T1b	Tumour >1 cm but ≤2 cm in greatest dimension[a]
T1c	Tumour >2 cm but ≤3 cm in greatest dimension[a]
T2	Tumour >3 cm but ≤5 cm or tumour with any of the following features:[c] – involves main bronchus regardless of distance from the carina but without involvement of the carina – invades visceral pleura – associated with atelectasis or obstructive pneumonitis that extends to the hilar region, involving part or all of the lung
T2a	Tumour >3 cm but ≤4 cm in greatest dimension
T2b	Tumour >4 cm but ≤5 cm in greatest dimension
T3	Tumour >5 cm but ≤7 cm in greatest dimension or associated with separate tumour nodule(s) in the same lobe as the primary tumour or directly invades any of the following structures: chest wall (including the parietal pleura and superior sulcus tumours), phrenic nerve, parietal pericardium
T4	Tumour >7 cm in greatest dimension or associated with separate tumour nodule(s) in a different ipsilateral lobe than that of the primary tumour or invades any of the following structures: diaphragm, mediastinum, heart, great vessels, trachea, recurrent laryngeal nerve, oesophagus, vertebral body, and carina
N: Regional lymph nodes involvement	
Nx	Regional lymph nodes cannot be assessed
N0	No regional lymph node metastasis
N1	Metastasis in ipsilateral peribronchial and/or ipsilateral hilar lymph nodes and intrapulmonary nodes, including involvement by direct extension
N2	Metastasis in ipsilateral mediastinal and/or subcarinal lymph node(s)
N3	Metastasis in contralateral mediastinal, contralateral hilar, ipsilateral or contralateral scalene, or supraclavicular lymph node(s)
M: Distant metastasis	
M0	No distant metastasis
M1	Distant metastasis present
M1a	Separate tumour nodule(s) in a contralateral lobe; tumour with pleural or pericardial nodule(s) or malignant pleural or pericardial effusion[d]
M1b	Single extrathoracic metastasise
M1c	Multiple extrathoracic metastases in one or more organs

Surgery is possible only till T 1-3 , N 0-1 , M0
Inoperable: T4, N2-N3, M1

INVESTIGATION

In all lung cancers generally, IOC is CT SCAN except for the following conditions where MRI is preferred:

- PAN COAST TUMOUR (Superior Sulcus Tumour) d/t Neurovascular bundle invasion (Brachial Plexus)
- Brain metastasis of Small Cell Lung Cancer

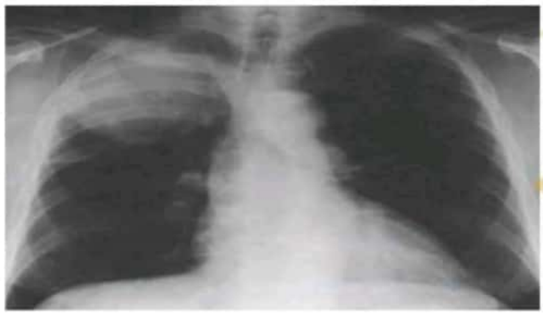


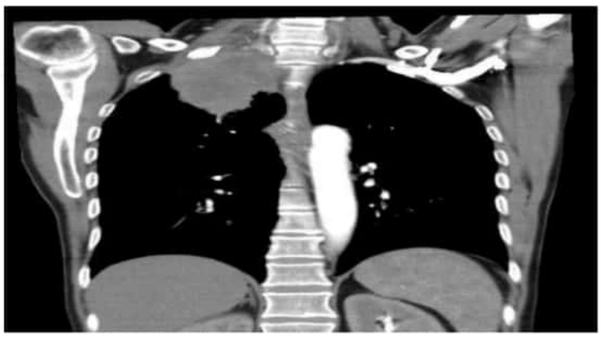
→ Staging of Lung Cancer: **CT-PET**
It's k/a **FDG-PET**

SUV- standardized uptake values used to denote the tumour cells uptake of metabolite in FDG-PET

Lung Cancer in CXR

- **Devoid of Air Bronchogram** *Except- Adenocarcinoma, Bronchoalveolar cancers.*
- Collapse
- Golden S sign
- Pancoast tumours- Rib destruction, vertebral involvement etc

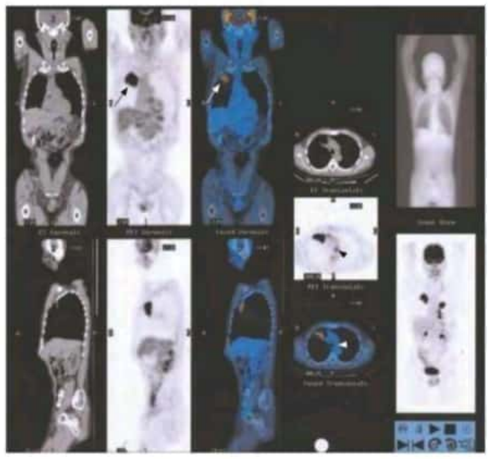




Pan-Coast Tumour in CT Scan
(though IOC is MRI for Pancoast)

Invasive Investigations:

- Bronchoscopy
- Endobronchial Ultrasound (EBUS) – can help in Transbronchial Needle Aspiration (TBNA) to get a tissue diagnosis.
- EUS-FNAC
- CT guided Biopsy
- Mediastinoscopy
- VATS (Video-assisted thoracoscopic surgery)



Peri-operative Mortality- THORACOSCORE
(9 parameter)

Table 1. Parameters of thoracoscore for predicting in-hospital mortality for patients requiring thoracic surgery.

Age (<55, 55-65, >65 years)
Sex
ASA classification (≤2, ≥3)
Performance status according to Zubrod scale (≤2, ≥3)
Severity of dyspnea according to Medical Research Council Scale (≤2, ≥3)
Priority of surgery (elective, urgent/emergency)
Extent of resection (pneumonectomy, other)
Diagnosis (malignant, benign)
Comorbidity score

ASA, American Society of Anesthesiologists.

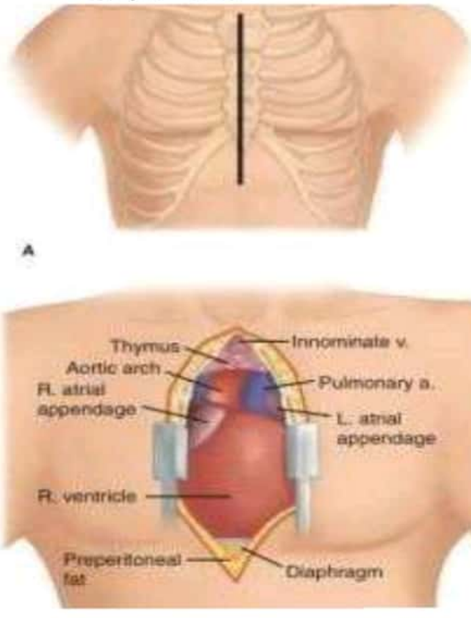
LUNG FUNCTION MARKERS:
a) FEV1
b) DLCO

Choice of Surgery: **Lobectomy**
If tumour is small: **Segmentectomy/wedge resection**
If Centrally placed tumour: **Pneumonectomy**

New- **Bronchoscopic Lung Resection** [Sleeve Lung Resection]

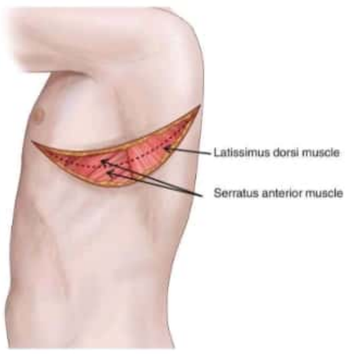
Median sternotomy
Done for –

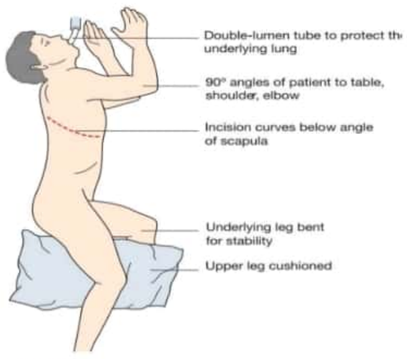
- Cardiac Surgery
- Mediastinal Mass



Postero-lateral Thoracotomy

Most commonly done for thorax




Double-lumen tube to protect th underlying lung
90° angles of patient to table, shoulder, elbow
Incision curves below angle of scapula
Underlying leg bent for stability
Upper leg cushioned

igure 55.19 Correct positioning for thoracotomy.

Anterolateral Thoracotomy

Done in Emergency situations

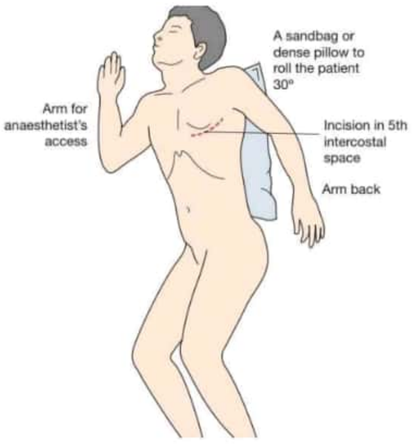
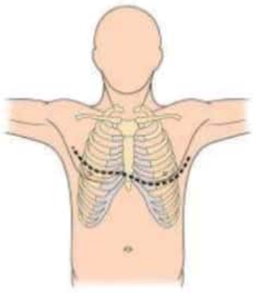

A sandbag or dense pillow to roll the patient 30°
Arm for anaesthetist's access
Incision in 5th intercostal space
Arm back

Figure 55.21 Emergency left anterior thoracotomy for access to the heart. This requires no special supports or devices.

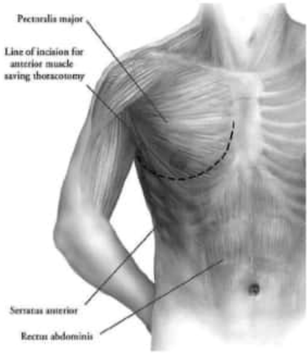
Clamshell thoracotomy



- Most of the time its extended from Left AL thoracotomy
- Provides better exposure to Both side lung with hilum, heart and descending aorta.
- Not good for Superior mediastinum, trachea, other great vessels.
- Avoid to cut breast tissue in female patient.

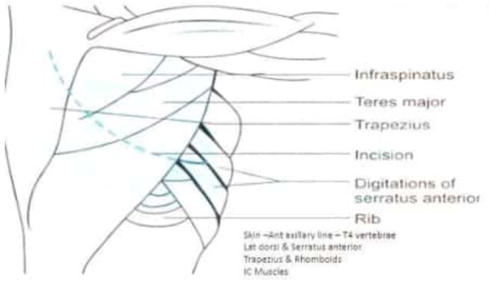
Structures pierced in Anterolateral Thoracotomy

- Skin 4 -5th IC space
- Subcutaneous tissue
- Pectoralis major
- Serratus anterior
- IC muscles



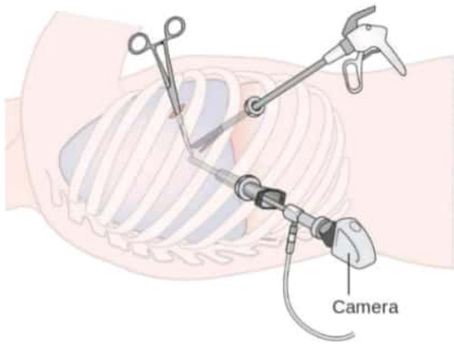
Structures pierced in Posterolateral Thoracotomy

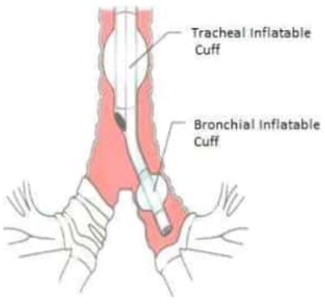
- Skin —Ant axillary line — T4 vertebrae
- Lat dorsi & Serratus anterior
- Trapezius & Rhomboids
- IC Muscles



VATS- Video Assisted Thoracoscopy

- Scope is but between the midaxillary line and the posterior axillary line in 7th or 8th ICS
- Hand instruments in 5th ICS
- I/L lung is deflated for this surgery using a double lumen tube for single lung ventilation.

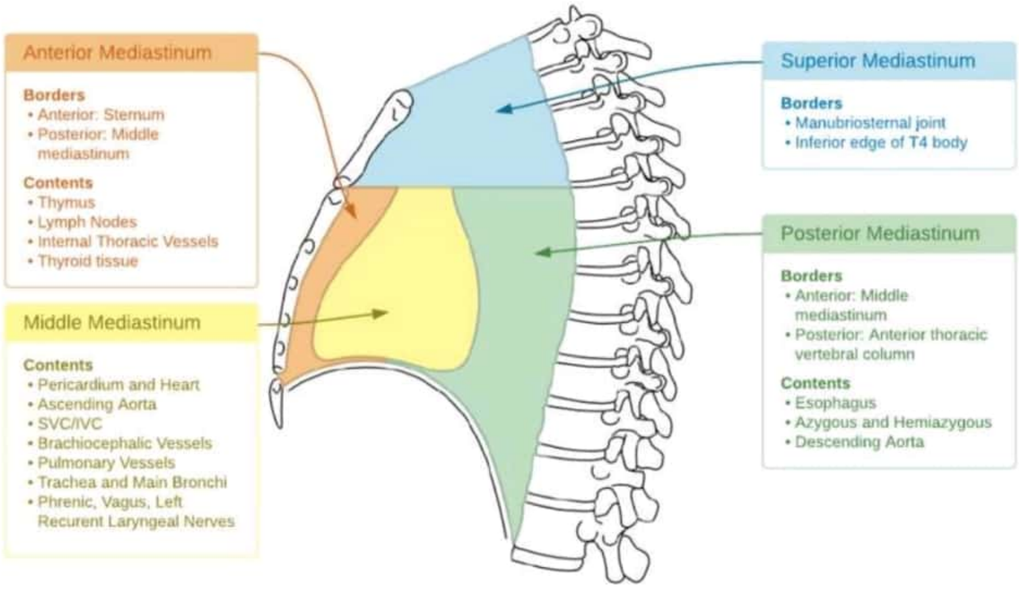




Mediastinal Masses

Case: A 25-year-old man undergoes a CXR to evaluate a shoulder injury that he sustained during a tennis match. A 4-cm anterior mediastinal mass is found and confirmed by CT scan. Which among the following is the most common cause of anterior mediastinal mass in adults?

ANATOMY



Anterior Mediastinum also contains superior & inferior **Sterno-pericardial ligaments**

Table 160-1. Usual Locations of the Common Primary Tumors and Cysts of the Mediastinum

Anterior Compartment	Visceral Compartment	Paravertebral Sulci
Thymoma	Enterogenous cyst	Neurilemoma (schwannoma)
Germ cell tumor	Lymphoma	Neurofibroma
Lymphoma	Pleuropericardial cyst	Malignant schwannoma
Lymphangioma	Mediastinal granuloma	Ganglioneuroma
Hemangioma	Lymphoid hamartoma	Ganglioneuroblastoma
Lipoma	Mesothelial cyst	Neuroblastoma
Fibroma	Neuroenteric cyst	Paraganglioma
Fibrosarcoma	Paraganglioma	Pheochromocytoma
Thymic cyst	Parathyroid cyst	Fibrosarcoma
Parathyroid adenoma	Pheochromocytoma	Lymphoma
Aberrant thyroid	Thoracic duct cyst	

Mediastinal tumors in adults

TUMOR TYPE	PERCENTAGE OF TOTAL	LOCATION
Neurogenic tumors	21	Posterior
Cysts	20	All
Thymomas	19	Anterior
Lymphomas	13	Anterior/middle
Germ cell tumors	11	Anterior
Mesenchymal tumors	7	All
Endocrine tumors	6	Anterior/middle

Mediastinal tumors in children

TUMOR TYPE	PERCENTAGE OF TOTAL	LOCATION
Neurogenic tumors	40	Posterior
Lymphomas	18	Anterior/middle
Cysts	18	All
Germ cell tumors	11	Anterior
Mesenchymal tumors	9	All
Thymomas	Rare	Anterior

NOTES NEXT DOOR

Signs and symptoms suggestive of various diagnoses in the setting of a mediastinal mass

DIAGNOSIS	HISTORY AND PHYSICAL FINDINGS	COMPARTMENT LOCATION OF MASS
Lymphoma	Night sweats, weight loss, fatigue, extrathoracic adenopathy, elevated erythrocyte sedimentation rate or C-reactive protein level, leukocytosis	Any compartment
Thymoma with myasthenia gravis	Fluctuating weakness, early fatigue, ptosis, diplopia	Anterior
Mediastinal granuloma	Dyspnea, wheezing, hemoptysis	Visceral (middle)
Germ cell tumor	Male gender, young age, testicular mass, elevated levels of human chorionic gonadotropin and/or α-fetoprotein	Anterior

TABLE 55.11 Masaoka thymoma staging system.

Stage	Description
I	Macroscopically completely encapsulated
	Microscopically no capsular invasion
II	Macroscopic invasion into surrounding fatty tissue or mediastinal pleura
	Microscopic invasion into the capsule
III	Macroscopic invasion into neighbouring organs (pericardium, great vessels, lungs)
IVA	Pleural or pericardial dissemination
IVB	Lymphogenous or haematogenous metastasis

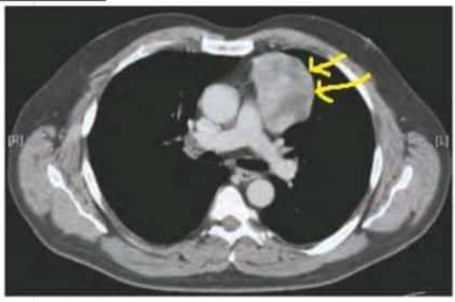
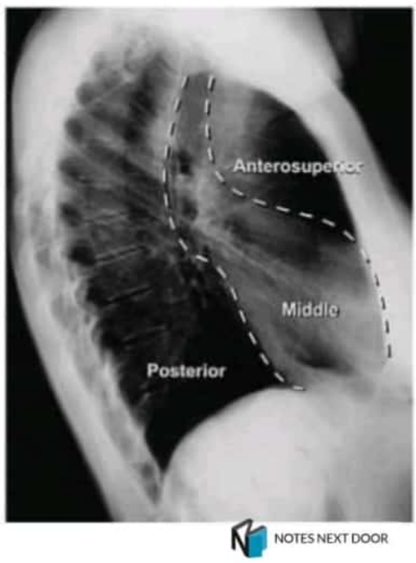


Figure 55.25 Computed tomography scan showing a thymoma presenting as a mediastinal mass.

Ptosis with CXR

a) Pancoast Tumor

b) Thymoma with Myasthenia Gravis



Treatment of Thymoma: **Thymectomy**

a. Median Sternotomy

b. VATS

NOTES NEXT DOOR

COPD

Case: A 35-year-old man works in ship yard seeks medical attention for breathlessness on exertion. He has never smoked cigarettes and has not been coughing. One sibling died of respiratory failure at 40 years of age. His three children are healthy. Physical examination reveals him to be tachypneic as he exhales through pursed lips. CXR is attached.

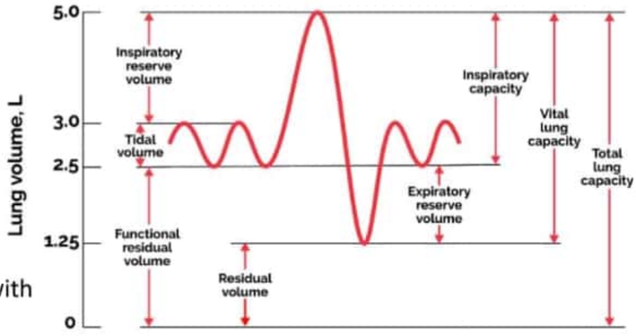
Xray findings: Hyperinflated lungs with tubular heart

Q. Expected results of the pulmonary function testing of the man described above would include
(A) increased FeV1
(B) increased total lung capacity
(C) reduced functional residual capacity
(D) increased vital capacity

Q. What is the possible diagnosis in the above clinical scenario ???
A. Asbestosis
B. Bronchiectasis
C. Primary pulmonary hypertension
D. Alpha 1 anti-trypsin deficiency

Q. What will be the best method to measure lung volumes in above case ???
A. Spirometry
B. Helium dilution method
C. Nitrogen wash out method
D. Body plethysmography

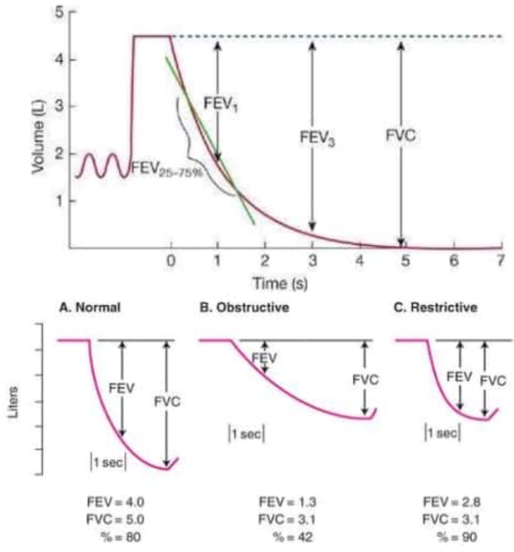
PHYSIOLOGY
SPIROMETRY



- Tidal Volume (TV)- Volume of air inspired OR expired.
  - Only volume to be same in males and females i.e. 500 mL.
- IRV- Volume of air that can be inspired over and above the TV with maximal effort.
  - Males = 3300mL
  - Females = 1900 mL
- ERV= Volume of air that can be expired over and above the TV with maximal effort.
  - Males= 1000 mL, Females = 700 mL
- Residual Volume- Males= 1200 mL; Females = 1100 mL
- Inspiratory Capacity = TV + IRV
- Expiratory Capacity = TV + ERV
- Functional Residual Capacity (FRC) = ERV + RV

- Vital Lung Capacity = IRV + TV + ERV
- Total Lung Capacity= IRV +TV+ ERV+ RV.
  - Males= 6000 mL
  - Females = 4200 mL

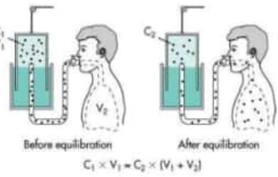
Timed Vital Capacity



- FEV1 = Air expired forcefully after a maximal inspiration in first **1 second.**
- Forced inspiration → Forced Expiration but to be done 'as fast as possible'
- Time is started to be measured from the moment of expiration begins.
  - FEV1 = 0.7 to 0.8 times the FVC
  - FEV2= 0.9 times the FVC
  - FEV3 = 0.97 to 0.99 times the FVC
- What cannot be measured by routine spirometry?
  - Residual volume
  - FRC
  - TLC
- FRC Can be measured by:
  1. Helium Dilution
  2. Nitrogen washout technique *(Different from Single Breath N2 Analysis- done for Anatomical Dead Space)*
  3. Whole Body Plethysmography
- Therefore, RV can be derived by subtracting ERV (from spirometry) from FRC

Helium Dilution Method (for TLC & FRC)*

- $[He]_{initial}$ = 10%
- V_S(initial) = 2 L
- $[He]_{final}$ = 5% (assume)
- Final Vol = $V_S + V_L$



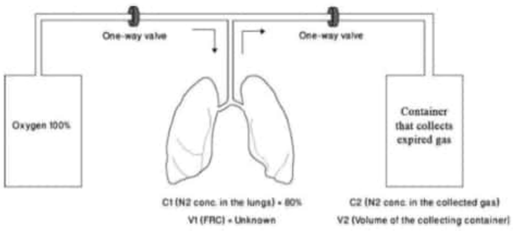
- C1xV1 = C2xV2
- $[He]_{initial} \times V_S = [He]_{final} \times (V_S + V_L)$

$$V_L = V_S \cdot \left(\frac{[He]_{initial}}{[He]_{final}} - 1\right)$$

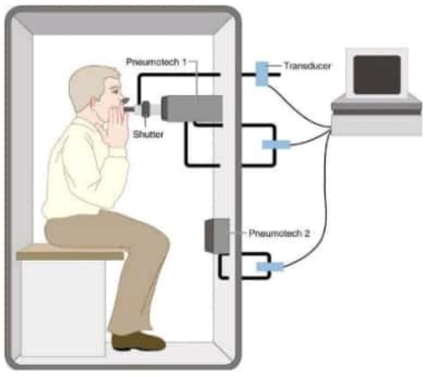
- Helium is used as it's an insoluble gas in blood.

Nitrogen washout technique

- Following a normal expiration (FRC), Pt. inspires 100% O2 and then expires it into spirometer (free of N2)
- over next few minutes (usually 6-7 min.), till all the N2 is washed out of the lungs.
- N2 conc. of spirometer is calculated followed by total vol.of AIR exhaled.
- As air has 80% N2 →so actual FRC calculated.


One-way valve
One-way valve
Oxygen 100%
Container that collects expired gas
C1 (N2 conc. in the lungs) = 80%
V1 (FRC) = Unknown
C2 (N2 conc. in the collected gas)
V2 (Volume of the collecting container)

Whole Body Plethysmography



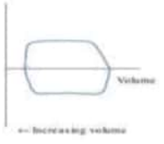
- Only technique which can calculate the air trapped in lungs
- Based on Boyle's Law (P1 X V1 = P2 X V2)
- Used to measure FRC
- At the end of the normal expiration the shutter is closed and then the patient is asked to inspire.
- This leads to equal loss of box volume resultantly increasing the box pressure
  - P1= Pre-inspiratory box pressure
  - V1= Pre-inspiratory box volume
  - P2= Post-inspiratory box pressure
  - V2= Post-inspiratory box pressure (=FRC)
  - P3= Pre-inspiratory lung pressure (mouth pressure)
  - P4 = Post-inspiratory lung pressure
  - ✓ P1 X V1 = P2 X (V1 - ΔV);
  - ✓ Thus ΔV can be calculated
  - ✓ V2 X P3 = (V2 + ΔV) X P4
  - ✓ This V2 can be calculated which is also the FRC
- After the inspiration the lung volume will increase and the lung pressure will decrease whereas the box volume will decrease and the box pressure will increase.

Radiology

- Investigations: HRCT more preferred than a CXR. HRCT can be done in both inspiratory and expiratory phase to detect air trapping
- HRCT can be employed to describe the pathological type of emphysema
  - Smoking- Centrilobular Emphysema
  - Alpha1 AT deficiency- Pan-lobular Emphysema.

Types of Lung Diseases

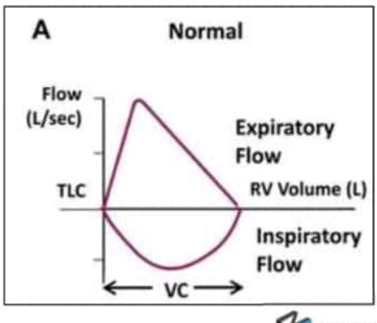
Case: A 65/M, chronic smoker is evaluated for progressive dyspnoea with a low pitched inspiratory and expiratory wheeze. His medical history is significant for an episode of necrotizing pancreatitis that resulted in ARDS and mechanical ventilation for 6 weeks prior to his recovery. The flow volume curve is shown in Figure. What is the most likely cause of the patient's symptoms?



Ans: Subglottic Stenosis

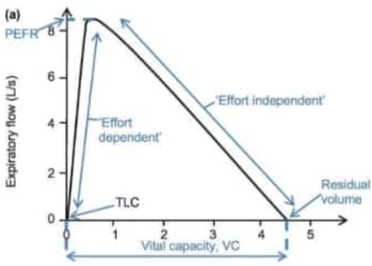
Physiology

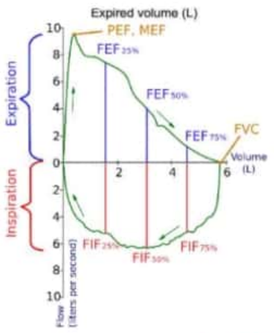
Flow Volume Loops



- Expiratory Flow Rate is calculated
- Starts from forced inspiration and then asked to forcefully expire as fast as possible.
- At start →TLC, At end→RV

- Normally the Expiratory flow rate is more in the beginnng (Effort dependent part), reaches a maximum point (PEFR) and then the Flow rate starts decreasing (Effort Independent part) due to DYNAMIC COMPRESSION OF AIRWAYS.
- At the end, the airways are completely collapsed, leaving behind the Residual Volume in the lungs.
- Also gives an idea about:
  - FVC = TLC- RV
  - MEFR (Mid expiratory Flow Rate)
    - Aka FEF 25-75%
    - Sensitive indicator of Obstructive Lung Ds



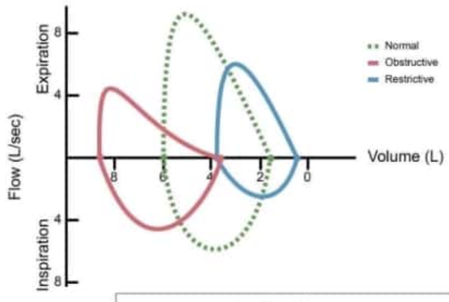


	Obstructive Lung Disease	Restrictive Lung Disease
TLC	N to ↑	↓
VC	↓ to N	↓
RV	↑	↓ (maybe increased in extra parenchymal)
FEV1/FVC	↓	N to ↑
PEFR	↓	N
MEFR	↓↓	N
Diffusing capacity	N except Emphysema	↓

✓ Volumes are more affected in Restrictive lung diseases.
✓ Rates are more affected in obstructive lung diseases.

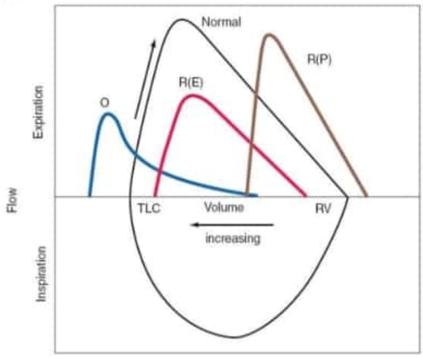
Obstructive Lung diseases:

- Increase in TLC
- Increase in RV
- PEFR is reduced
- "Scooped out" curve



Restrictive Lung diseases:

- Decrease in TLC
- Decrease in RV (except extra-parenchymal)
- PEFR is increased
- Rates are the ones to be more affected
- Tall and Narrow curve

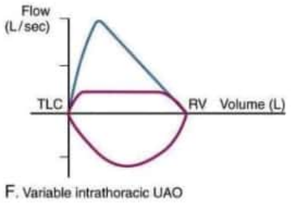


Extra-parenchymal Restrictive:

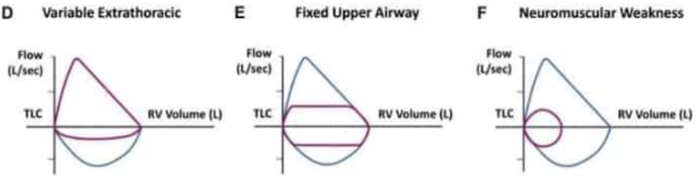
- Marked as R (E) in this graph → →
- Normal or Increased RV
- Curve LIES WITHIN the normal curve actually.

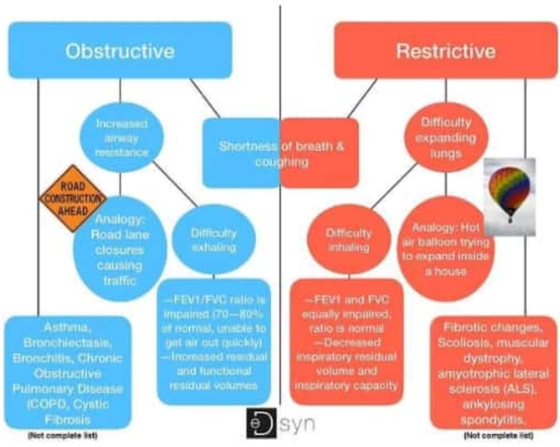
Airway Obstruction:

- *Extra-thoracic*: Lies above 6th tracheal ring.
- *Intra-thoracic*: Lies below the 6th Tracheal ring.



F. Variable intrathoracic UAO




Obstructive
Restrictive
Increased airway resistance
Shortness of breath & coughing
Difficulty expanding lungs
ROAD CONSTRUCTION AHEAD
Analogy: Road lane closures causing traffic
Difficulty exhaling
Difficulty inhaling
Analogy: Hot air balloon trying to expand inside a house
—FEV1/FVC ratio is impaired (70—80% of normal, unable to get air out quickly)
—Increased residual and functional residual volumes
—FEV1 and FVC equally impaired, ratio is normal
—Decreased inspiratory residual volume and inspiratory capacity
Asthma, Bronchiectasis, Bronchitis, Chronic Obstructive Pulmonary Disease (COPD, Cystic Fibrosis
(Not complete list)
Fibrotic changes, Scoliosis, muscular dystrophy, amyotrophic lateral sclerosis (ALS), ankylosing spondylitis
(Not complete list)
syn

Lung Physiology

Case: **A 24-year-old woman is brought to the emergency room after attempting suicide with an overdose of heroin. She is obtained and has a respiratory rate of 6 breaths/min. She is hypotensive with a blood pressure of 92/60 mmHg and a heart rate of 80 beats/min. The oxygen saturation is 70% on room air. ABG: pH 7.09, PaCO2 80 mmHg (HYPERCAPNIA), PaO2 42 mmHg (HYPOXEMIA)**

a. Which of the following statements is true regarding the patient's arterial blood gas?

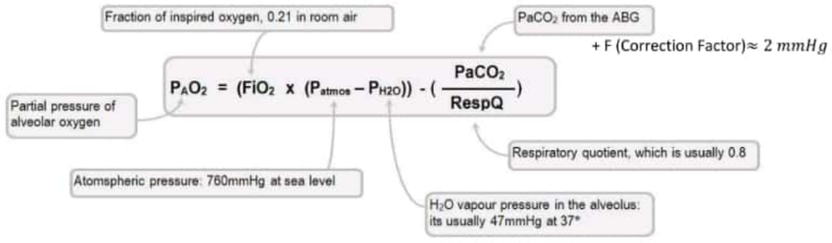
A. The patient is hypoxic due to hypoventilation with an increased A – a (alveolar-arterial) gradient.

B. The patient is hypoxic due to hypoventilation with a normal A – a gradient.

C. The patient is hypoxic due to shunt with an increased A – a gradient.

D. The patient is hypoxic due to ventilation-perfusion (V• /Q•) mismatch with an increased A – a gradient

Physiology- Alveolar Gas Equation


Fraction of inspired oxygen, 0.21 in room air
PaCO2 from the ABG
+ F (Correction Factor)≈ 2 mmHg
PAO2 = (FiO2 x (Patmos – PH2O)) - (PaCO2 / RespQ)
Partial pressure of alveolar oxygen
Respiratory quotient, which is usually 0.8
Atomspheric pressure: 760mmHg at sea level
H2O vapour pressure in the alveolus: its usually 47mmHg at 37°

For FiO2 of 21%, this P_aO_2 comes out to be 100 mm of Hg

- Therefore, when the air travels from the atmosphere to the alveoli, the partial pressure of oxygen decreases due to:
  - Addition of water vapours (humidification of inspired air)
  - Addition of carbon dioxide (due to gas exchange from blood)

A-a gradient = P_AO_2 – P_aO_2
alveolar gas equation; ABG

Alveolar-arterial oxygen gradient P(A-a)O2

- The difference between the amount of O2 that enters the alveoli and the amount that gets to the blood stream
- Non-invasive bedside tool used to quantitate the efficiency of oxygen loading
- An increase in P(A-a)O2 indicates an increase in shunt
- PAO2 – PaO2 = P(A-a)O2

- This gradient is due to VENOUS ADMIXTURE OF BLOOD into the Arterial Supply.
  - This is because of **'Physiological Shunting'** due to the BRONCHIAL, PLEURAL, and THEBESIAN veins which flow into the Pulmonary veins that then flow into the Left Atrium, thus bypassing the lungs.
- Normally A-a Oxygen gradient is 5-15 mm of Hg
- Increased in conditions like Right to Left Shunt, or a V-Q Mismatch.
- In high altitudes- the both the alveolar and the corresponding arterial oxygen will decrease, keeping the gradient in normal limits.

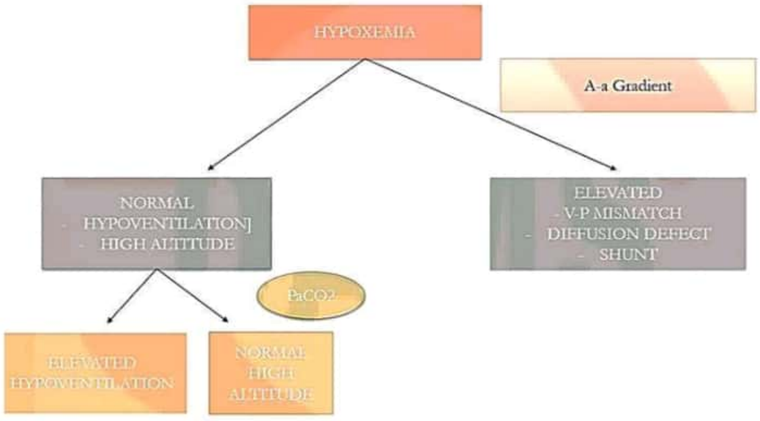
Alveolar-Arterial P_{O_2} Difference

Alveolar CO_2 and arterial CO_2 are equal. The same is not true for alveolar and arterial O_2. Even in normal individuals, alveolar O_2 is slightly greater than arterial O_2. The difference between alveolar O_2 (PAO_2) and arterial P_{O_2} (Pa_{O_2}) is called the alveolar-arterial P_{O_2} difference (AaD_{O_2}). An increased in AaD_{O_2} is a hallmark of abnormal O_2 exchange. This small difference is not caused by "imperfect" gas exchange, but by the small number of veins that bypass the lung and empty directly into the arterial circulation. The thebesian vessels of the left ventricular myocardium drain directly into the left ventricle (rather than into the coronary sinus in the right atrium), and some bronchial and mediastinal veins drain into the pulmonary veins. This results in venous admixture and a decrease in arterial P_{O_2}. (This is an example of an anatomic shunt; see later.) Approximately 2% to 3% of cardiac output is **shunted** in this way.

Solution the to Question-

- PAO2 = ((Patm – PH2O)*(FIO2)) – (PaCO2/R),
- PAO2 =(760 –47)(0.21) – (80/0.8) = 150 –100
- In this patient, the calculated PAO2 is 50.
- Thus, the A – a gradient is 8 mmHg (normal value <15 mmHg) and is normal.

Mechanism and approach to hypoxemia



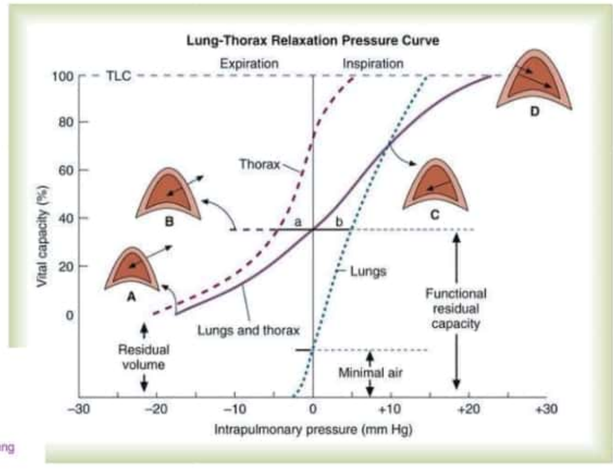
TYPES of Respiratory Failure

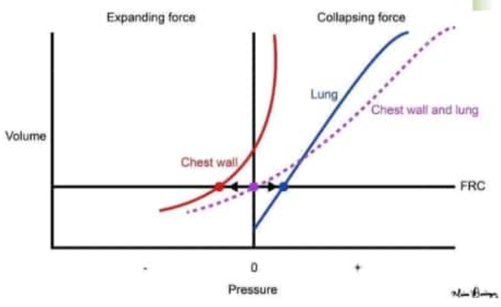
Physiologic Classification

	Type 1 Hypoxemic	Type 2 Hypercarbic	Type 3 Post-op	Type 4 Shock
Mechanism	Shunt	↓Va	Atelectasis	↓Cardiac Output
Etiology	Airspace Flooding	Increased Respiratory load, Decreased ventilatory drive	Decreased FRC and increased Closing Volume	Decreased FRC and increased Closing Volume
Clinical Setting	Water, Blood or Pus filling alveoli	CNS depression, Bronchospasm, Stiff respiratory system, respiratory muscle failure	Abdominal surgery, poor insp effort, obesity	Sepsis, MI, acute hemorrhage

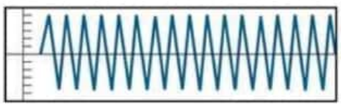
Pressure Volume Curves for Chest wall, Lung and combined chest wall & lung

- Elastic recoil of lung tends to collapse it.
- Elastic recoil of chest wall tends to expand it.
- Oppsite forces create a sub-atmospheric pressure inside the pleura.

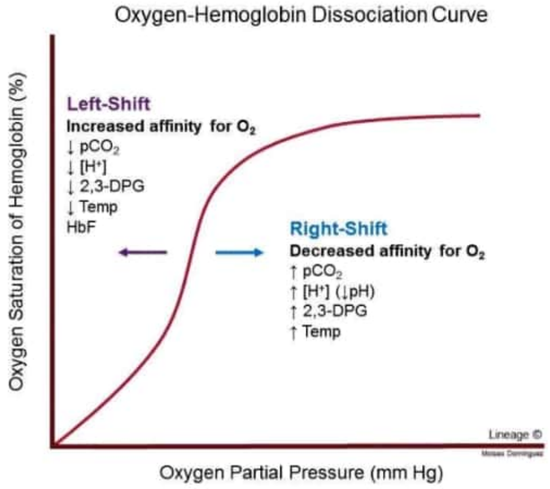




- **Kussmaul Breathing**
  - Ketoacidosis
  - Uremia
  - Sepsis
  - Methanol poisoning
  - Aldehyde poisoning
  - Salicylate poisoning
  - Lactic Acidosis

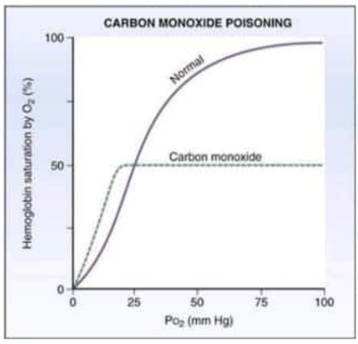
Cheyne-Stokes Respiration	Tidal volume (V_T) with a progressive shallow-deep-shallow cycle (30 sec–2 min)	CVA, trauma, or masses involving a diffuse area of the forebrain, carbon monoxide poisoning, metabolic encephalopathy, altitude sickness, opiate effect
Biot's (Cluster) Respiration	Periods of rapid respirations of nearly equal depth or V_T followed by regular periods of apnea	CVA or trauma involving the medulla or lower pons, mass effect on the medulla secondary to uncal or tentorial herniation, opiate effect
Kussmaul Respiration/ Hyperpnea	 Hyperventilation characterized by a consistent and deep respiratory pattern; the RR of patients with hyperpnea may be normal or rapid with increased V_T	Severe metabolic acidosis; Kussmaul respiration in diabetic ketoacidosis starts with a rapid and shallow pattern and later becomes rapid and deep
Apneustic Respiration	Prolonged inspiratory phase followed by prolonged expiratory phases believed to be apneic phases	CVA, trauma, or mass effect involving the pons
Ataxic Respiration	Erratic breathing pattern with irregular pauses and increasing episodes of apnea (progresses to agonal respiration)	CVA or trauma involving the medulla (indicates a very poor prognosis)

- Sigmoid shape of curve due to phenomenon of **Positive Co-operativity** (which is the progressively increase of oxygen affinity of haemoglobin with attachment of each of the heme)
- P50 is the point at which the haemoglobin is 50% saturated; corresponds to PO2 of 25-27mmHg or 3.7 KPa



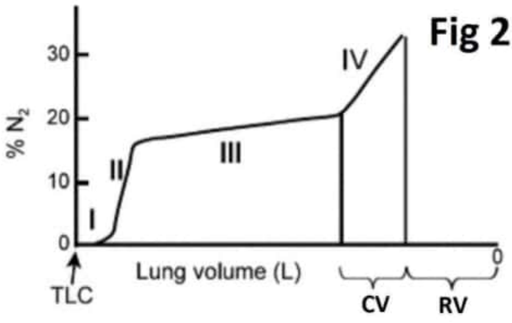
Carbon Monoxide poisoning

- Decreases the saturation (flattening of the curve) due to irreversible attachment to heme.
- Also shifts the curve to left, because of causing the decreased in 2,3 DPG, indicating increased oxygen affinity.



Measurement of Anatomical Dad Space by Single Breath Nitrogen Analysis (Fowler's Method)

- Ask the subject to inhale 100% oxygen in mid-inspiration. Then while the subject breathes out, the nitrogen content of the expired air is measured and plotted.
- I: Pure dead space air (because first the subject will breath out air in the major airways which is 100% oxygen)
- II: Dead Space + Alveolar Air
- III: Alveolar Air



- **ANATOMICAL Dead Space = Vol. I + (Vol II/2)**
- **PHYSIOLOGICAL Dead Space = Anatomical Dead Space + Wasted Ventilation (V/Q >1.0) (aka TOTAL dead space)**

- **BOHR's Equation**

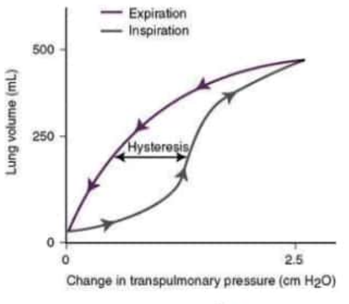
$$\frac{V_D}{V_T} = \frac{(PACO_2 - P_ECO_2)}{PACO_2}$$

which is nearly about 30% normally

  - V_D = Dead Space Volume
  - V_T = Tidal volume
  - PACO2 = CO2 concentration in Alveoli/Arterial
  - PECO2= CO2 Concentration in expired air

Hysteresis Loop

- Flatter the curve, lesser the compliance
- Steeper the curve, more the compliance
- Compliance= Change in volume/ Unit change in pressure
- Cause of Hysteresis: Surface tension of surfactant
- Compliance is ↑ in expiration, because with expiration the alveolar size ↓, which ↑es the surfactant concentration, which ↓es the surface tension, ↑sing the compliance.

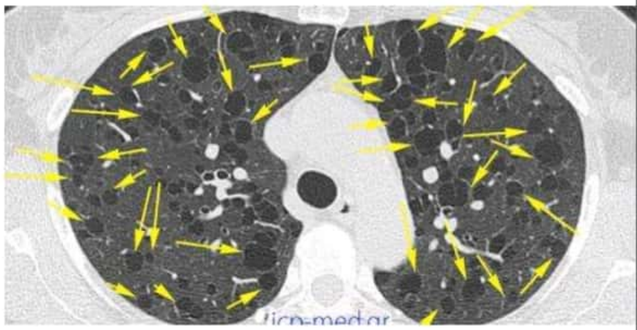


NOTES NEXT DOOR

CYSTIC LUNG DISEASES

Case: A 35-year-old female was admitted with acute dyspnoea, 12 months after the birth of her second child. During the past 6 months, she has suffered from mild dyspnea despite the fact that she has never smoked. Chest radiography revealed a unilateral pneumothorax, which was treated appropriately. The follow-up CT scan is shown below.

HRCT Findings: Uniform cystic spaces in B/L Lungs.



Causes of Recurrent Pneumothorax:

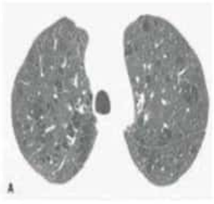
Male(Smoker) : Langerhans cell histiocytosis

Female: (specially repro age group) Lymphanhgioleiyomyomatosis

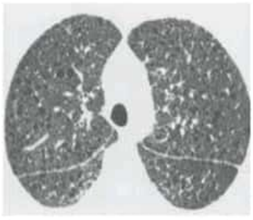
DISEASE	PULMONARY LANGERHANS' CELL HISTIOCYTOSIS	PULMONARY LYMPHANGIOLEIOMYOMATOSIS
MECHANISM	Aberrant accumulation of Langerhans' cells in various organs →nodular inflammatory lesions →cavitating destruction of the lung →may evolve to thick- then thin walled cysts	proliferation of atypical smooth muscle along lymphatics in the lung and abdomen -Destruction of alveoli → cyst formation - Rupture of lymphatics of the pleura → Chylothorax - AngioMyolipomas (AMLs) of the kidney
Clinical presentation	Young Male Smokers →Pneumothorax	Women of reproductive age →Pneumothorax
CT chest	Centrilobular nodules **B/L upper lobe** predominance cysts. Not very uniform.	**Diffuse** Uniform size cysts; pleural effusion
RX	Smoking Cessation, steroids. Lung transplant	Immunosuppressive agent sirolimus Lung transplant

LCH: is also a differential for Miliary Mottling.

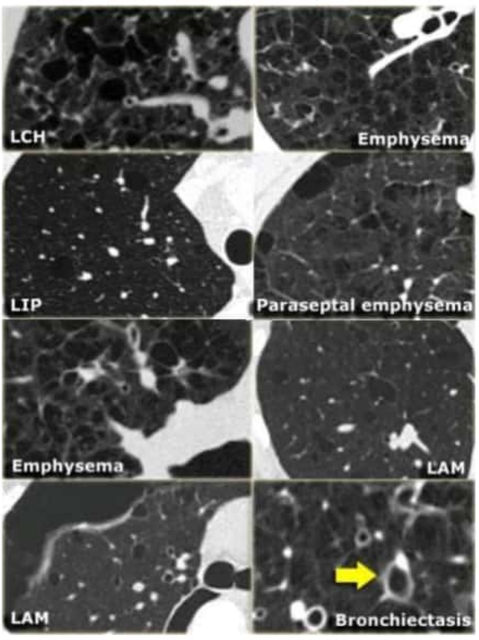
LAM: shows recurrent chylothorax in a young female; believed to be a PEComa, associated with Tuberous Sclerosis complex.



LCH with upper lobe predominance



LAM Cysts are rounder and more regular in size than those seen in LCH

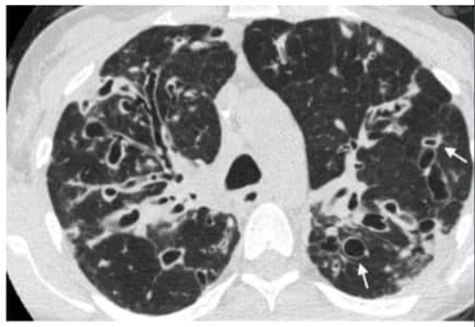


	LAM	PLCH	LIP	BHD	Amyloidosis
Age at diagnosis	Adults	Younger adults, 3rd and 4th decades	Broad range	Adults	Adults
Gender	Nearly all women	No predominance	No predominance	No predominance	No predominance
Relevant history	Pneumothorax in majority; some have underlying TSC	Nearly all have smoking history; pneumothorax in 15%	Some have underlying CTD, immunodeficiency, etc.	Pneumothorax in 15%–25%; family history of BHD, renal tumor or pneumothorax	Some have systemic amyloidosis or underlying disease, e.g., CTD
Extrapulmonary manifestations	Renal angiomyolipoma, chylous ascites, stigmata of TSC	Bone lesions, diabetes insipidus	Signs of underlying disease, e.g., CTD, HIV infection, etc.	Benign skin tumors, renal neoplasms	Signs of underlying systemic amyloidosis, CTD, etc.
Laboratory testing	Elevated serum VEGF-D level, genetic testing (TSC)	NA	Dysproteinemia (polyclonal)	Genetic testing (*FLCN* gene mutations)	Dysproteinemia (monoclonal)
HRCT findings	Many round cysts, mostly 2 mm to 2 cm in size, with normal intervening lung parenchyma; diffuse distribution	Irregular cysts, often with nodules and architectural distortion of intervening parenchyma; relative sparing of lung bases	Cysts of varying sizes and shapes, often with ground-glass opacities, nodules, septal thickening and lymphadenopathy	Cysts of varying sizes and shapes; more prominent in lower lobes	Scattered cysts of varying sizes, often with nodules

BHD, Birt-Hogg-Dubé syndrome; CTD, connective tissue disease; HIV, human immunodeficiency virus; HRCT, high-resolution CT; LAM, lymphangioleiomyomatosis; LIP, lymphoid interstitial pneumonia; NA, not available; PLCH, pulmonary Langerhans' cell histiocytosis; TSC, tuberous sclerosis complex; VEGF-D, vascular endothelial growth factor-D.

ABPA – Allergic Broncho-Pulmonary Aspergillosis

Case: A 37-year-old patient presents with adult-onset asthma. Due to increased production of brownish sputum production, and perihilar and upper lobe opacities on a conventional chest radiograph. A CT image is shown below.



Suspect ABPA when you find Asthma or Cystic Fibrosis with Bronchiectasis

Q. Which of the following statements concerning a suspected underlying disorder is correct:

a) Fungal invasion of the bronchial mucosa is typically found in bronchoscopic biopsy specimens.

b) Total serum IgE and blood eosinophils are typically normal.

c) A skin prick test to *A. fumigatus* is usually positive

d) Immunosuppressive therapy should be started with a combined regimen of glucocorticoids & cyclophosphamide or a monoclonal antibody against CD20 receptor

Q. Diagnostic features of above condition include all of the following except.

a. CXR infiltrates.

b. Peripheral eosinophilia.

c. H/O Asthma or cystic fibrosis.

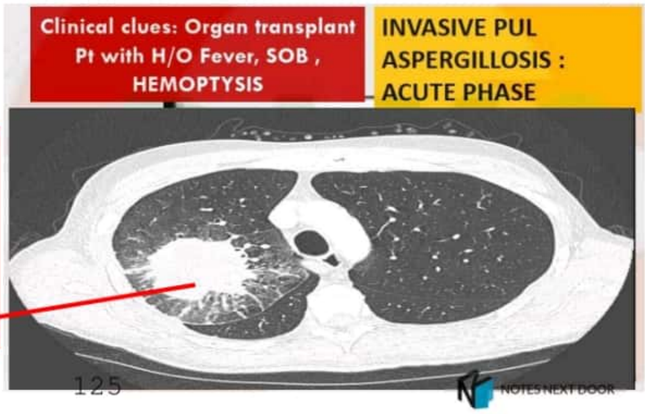
d. Occurrence in patients with old cavitatory lesion

CRITERIA FOR ABPA :

- Predisposing conditions : Bronchial asthma, cystic fibrosis
- SKIN test positive to Aspergillus fumigatus
- Elevated total IgE levels (> 1000 IU/mL)
- Presence of IgE and IgG antibodies against Aspergillus fumigatus
- Radiographic pulmonary opacities consistent with ABPA†
- Peripheral Eosinophilia

- **Gloved Finger appearance also seen in ABPA due to bronchial plugging.**

CT HALO sign: Central consolidation with surrounding haziness not obscuring the broncho- vascular architecture k/a GROUND GLASS APPEARANCE.

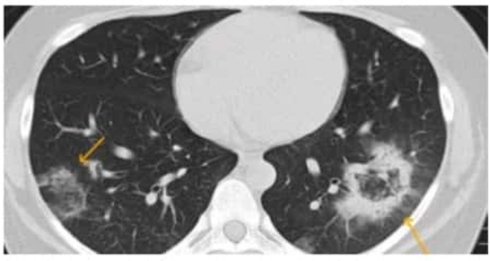

Clinical clues: Organ transplant Pt with H/O Fever, SOB , HEMOPTYSIS
INVASIVE PUL ASPERGILLOSIS : ACUTE PHASE
125
NOTES NEXT DOOR

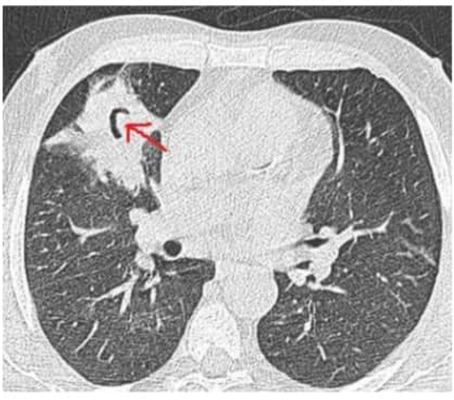
ATOLL SIGN/ REVERSE HALO SIGN

Central Ground glass with peripheral consolidation

Seen in

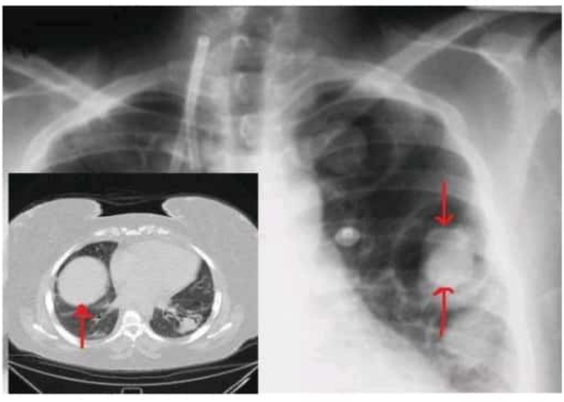
1. Cryptogenic **organizing pneumonia** (COP)
2. COVID-19





Resolving Invasive Pulmonary Aspergillosis

Arrow: Air Crescent Sign



Monod's sign: ASPERGILLOMA seen in old cases of Pulm TB

ASPERGILLOMA: The ball changes its position in in cavity- in the decubitus position.

If it does not move- it could be a Intracavitatory Tumour.

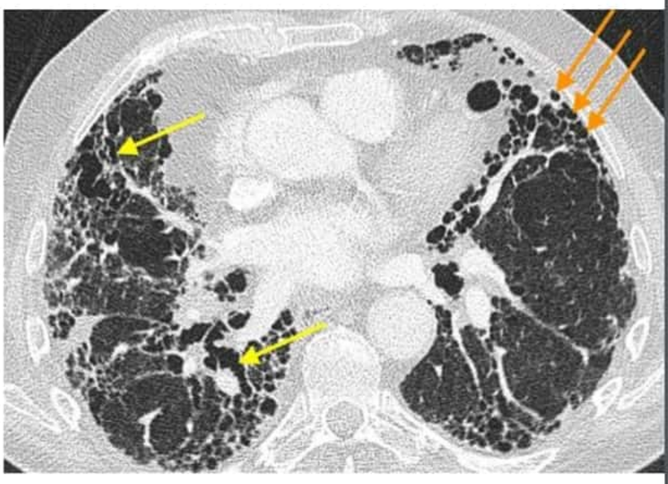
Interstitial Lung Diseases

Case: A 60-year-old female is referred for dyspnoea on exertion and chronic cough. Her dyspnoea and cough have worsened continuously during the past 12 months. Pulmonary function testing reveals an FVC of 72% predicted, FEV1 of 80% predicted and a DLCO of 38% predicted. A representative slice of the chest CT is shown below. Open-lung biopsy reveals randomly distributed foci of scarring with fibroblasts surrounded by normal lung parenchyma.

HRCT Finding:

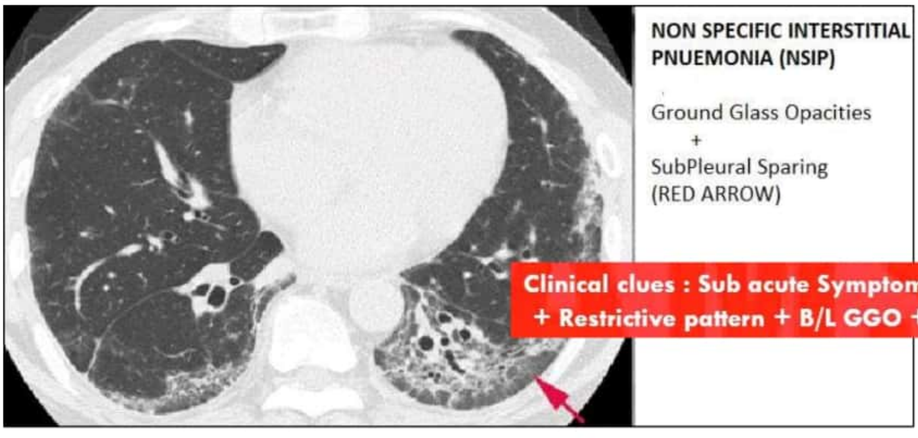
Yellow Arrow: **Tractional Bronchiectasis**

Orange Arrow: **Honeycombing**



Dx: Usual Interstitial Pneumonia (UIP)

Rx: Anti-Fibrotic Drugs
1) PIRFENIDONE
2) NINTEDANIB


NON SPECIFIC INTERSTITIAL PNUEMONIA (NSIP)
Ground Glass Opacities
+
SubPleural Sparing
(RED ARROW)
Clinical clues : Sub acute Symptoms + Restrictive pattern + B/L GGO +

NSIP:

- Middle Age patient
- Good Prognosis (as Honeycombing has not developed and the lung architecture is preserved.
- Rx- Steroids

Pulmonary Alveolar Proteinosis

Case: A 50-year-old female with an unremarkable previous medical history reports progressive dyspnoea. The chest CT is shown below. Which one of the following is the most appropriate diagnostic evaluation to perform next?

HRCT: Ground Glass opacities with septal thickening (k/a **CRAZY PAVEMENT PATTERN**)

Dx: **Milky White** Broncho-Alveolar Lavage

- Abnormal accumulation of pulmonary surfactant in the alveolar space
- MC : Primary form : anti-GM-CSF antibody production
- Secondary form : Silicosis, Hematological malignancies

Lung Collapse

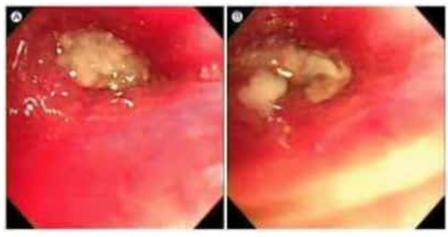
Case: A 68/M underwent an open cholecystectomy 48 hr ago and now c/o SOB and cough. He has tachycardia and tachypnea, Spo2 : 90% with room air. No H/o chest pain / lower limb tenderness / hemoptysis. Chest examination reveals decreased movements of chest wall right side and breath sounds not audible. CXR is shown.

CXR: Right lung field is opaque along with right shift of mediastinum. Therefore, there is loss of volume of right lung. Suggestive of Right Lung Collapse.

Other Probable Dx: Foreign body aspiration (if in a paediatric age group case)

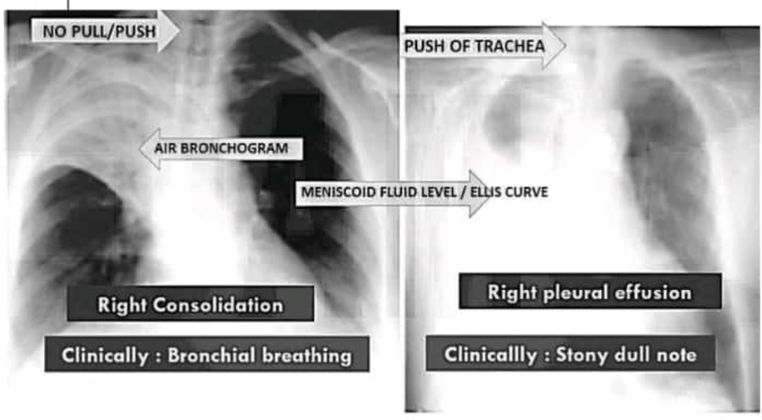
Etiology of Collapse: Post-operative retention of secretions.

Rx: Bedside- Bronchoscopy and removal



OPACIFIED HEMITHORAX

Atelactasis / collapse
Pleural effusion
Extensive consolidation



PULMONARY EMBOLISM

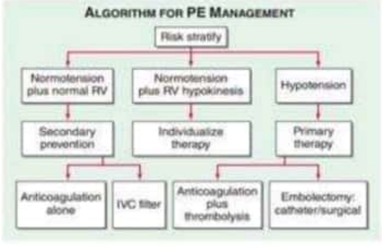
Case: A 27/ F junior doctor presents to ED with pleuritic chest pain and sob. Her ABG shows PaO2. She uses OCP. Her vital signs are: blood pressure 86/44 mmHg, heart rate 130/ min, respiratory rate 30 /min, SaO2 85% on room air. Her ECHO reveals RV hypokinesia. CT chest is shown.

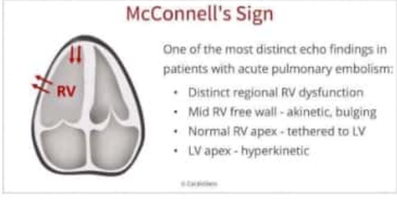
What is the most appropriate therapy?

a. Anti-coagulation alone
b. Anti-coagulation plus thrombolysis
c. Diuretic plus anti coagulation
d. Surgical embolectomy

CT Angiography: Saddle shaped filling defect in pulmonary artery.

IOC:- CT Angiography (CXR is usually normal)




McConnell's Sign
RV
One of the most distinct echo findings in patients with acute pulmonary embolism:
Distinct regional RV dysfunction
Mid RV free wall - akinetic, bulging
Normal RV apex - tethered to LV
LV apex - hyperkinetic

- IVC filters allows blood to pass through while large emboli from the pelvis or lower extremities are blocked or fragmented before reaching the lung.
- The indications for inferior vena cava filter placement are :
  - active bleeding precluding anticoagulation,
  - recurrent deep venous thrombosis on adequate anticoagulation.

LUNG ABSCESS

Case: A 65 yr Old male with previous history of a CVA presents with 3-week history of SOB, a cough productive of foul-smelling sputum, intermittent fevers and fevers and wt loss. He has been prescribed several antibiotics but no significant improvement. His CXR is given below.

Dx: Lung Abscess

Which one of the following mostly likely diagnosis

A. Pulmonary TB
B. Bronchogenic Carcinoma
C. Lung abscess
D.Hydropneumothorax

What is the most appropriate treatment ?

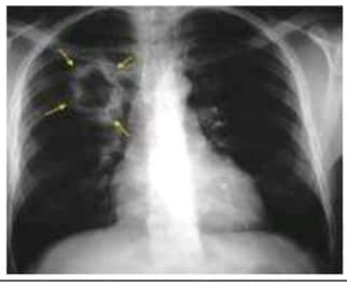
a. Intercostal drainage
b. Cisplatin plus gemcitabine
c. Anti tubercular therapy
d. Clindamycin

Primary Lung Abscess: More common
Due to aspiration, M/C Organisms are Oral anaerobes

Secondary Lung Abscess: Due to pre-existing lung diseases. Organism- Staph aureus, psueodomonas

BRONCHOGENIC CARCINOMA

Case: A 60 / M , HEAVY SMOKER bronchogenic carcinoma presenting with polyuria, pain abdomen, nausea, vomiting, altered sensorium with the below chest xray .The histological variant can be mostly-



Ans: Squamous cell carcinoma (causing Hypercalcemia)

Chest Xray: Cavitating mass with a thick irregular/nodular walls

PARANEOPLASTIC SYNDROMES IN LUNGCANCER :

SMALL CELL LUNG CANCER
- Hyponatremia : SIADH
- Hypokalemia : Ectopic ACTH
- Hypocalcemia : calcitonin

SQUAMOUS CELL CANCER : Hypercalcemia

ADENO Ca : Hematological Paraneoplastic syndromes

4 C's of Squamous cell LUNG CANCER

- Centrally located
- Cigarette smoking- highest association
- Cavitating lesion
- hyperCalcemia

M/C Lung Cancer: Adenocarcinoma

More Central: Squamous and small-cell carcinoma
More Peripheral: Adenocarcinoma and acinar cell carcinoma

As an oncologist, you are considering treatment options for your patients with lung cancer, including small-molecule therapy targeting the epidermal growth factor receptor (EGFR). Which of the following patients is most likely to have an EGFR mutation?

A. A 23-year-old man with a hamartoma

B. A 33-year-old woman with a carcinoid tumor

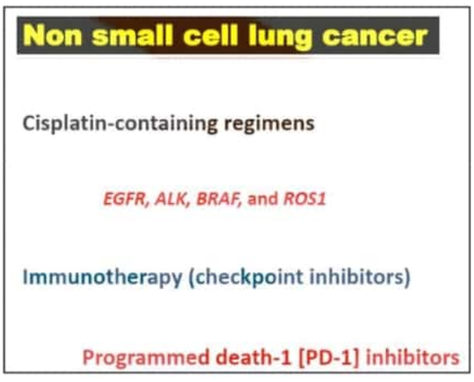
C. A 45-year-old woman who has never smoked with an adenocarcinoma

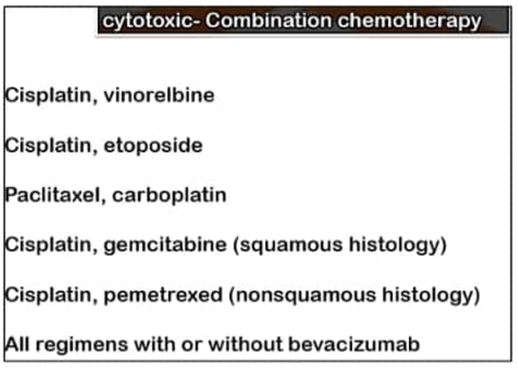
D. A 56-year-old man with a 100-pack-year history of tobacco use with small-cell lung carcinoma

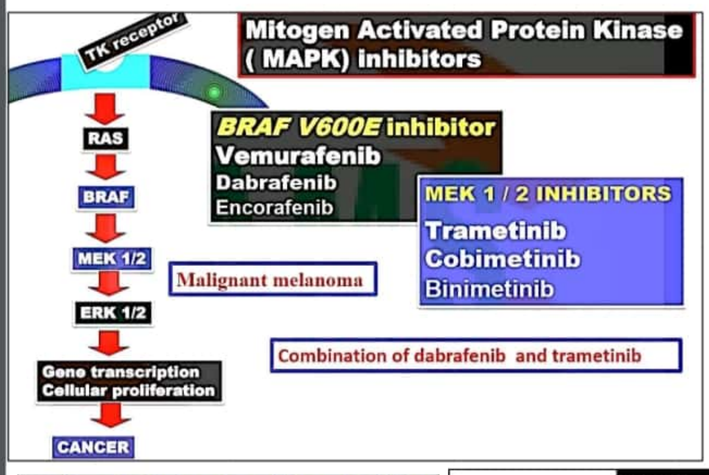
Immunotherapy approved in treatment of Lung cancer

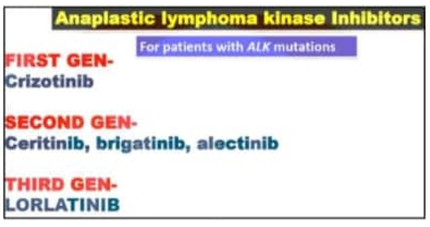
1. Mepolizumab 2.Nivolumab 3. Pembrolizumab 4. Reslizumab 5.Omalizumab

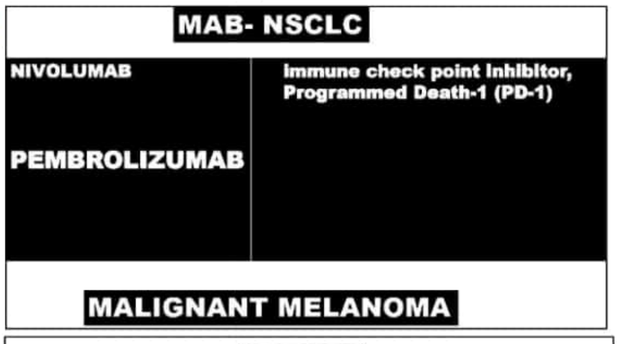
A. All B. only 1234 **C. Only 2 & 3** D. Only 123

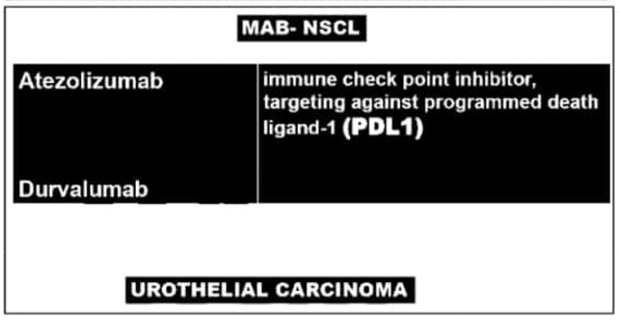


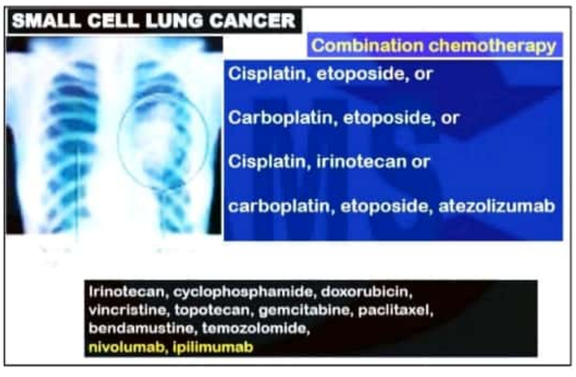



TK receptor
Mitogen Activated Protein Kinase (MAPK) inhibitors
RAS
BRAF
MEK 1/2
ERK 1/2
Gene transcription
Cellular proliferation
CANCER
BRAF V600E inhibitor
Vemurafenib
Dabrafenib
Encorafenib
MEK 1 / 2 INHIBITORS
Trametinib
Cobimetinib
Binimetinib
Malignant melanoma
Combination of dabrafenib and trametinib


Anaplastic lymphoma kinase Inhibitors
For patients with ALK mutations
FIRST GEN-
Crizotinib
SECOND GEN-
Ceritinib, brigatinib, alectinib
THIRD GEN-
LORLATINIB


MAB- NSCLC
NIVOLUMAB
PEMBROLIZUMAB
immune check point inhibitor, Programmed Death-1 (PD-1)
MALIGNANT MELANOMA


MAB- NSCL
Atezolizumab
Durvalumab
immune check point inhibitor, targeting against programmed death ligand-1 (PDL1)
UROTHELIAL CARCINOMA


SMALL CELL LUNG CANCER
Combination chemotherapy
Cisplatin, etoposide, or
Carboplatin, etoposide, or
Cisplatin, irinotecan or
carboplatin, etoposide, atezolizumab
Irinotecan, cyclophosphamide, doxorubicin, vincristine, topotecan, gemcitabine, paclitaxel, bendamustine, temozolomide, nivolumab, ipilimumab

MESOTHELIOMA

Combination chemotherapy

Cisplatin, pemetrexed, bevacizumab or

Carboplatin, pemetrexed, with or without bevacizumab or

Gemcitabine, cisplatin

Vinorelbine, nivolumab with or without ipilimumab, pembrolizumab

MAB- BRONCHIAL ASTHMA

OMALIZUMAB – Ig E

DUPILUMAB IL-4

ATOPIC DERMATITIS

MEPOLIZUMAB-IL5

RESLIZUMAB-IL5

BENRALIZUMAB- IL5

TRALOKINUMAB IL-13

LEBRIKIZUMAB -IL13

CYSTIC FIBROSIS

- IVACAFTOR
- LUMACAFTOR
- TEZACAFTOR

INHALATIONAL ANTHRAX

- RAXIBACUMAB
- OBILTOXAXIMAB

PNEUMONIA

Case: A 68/M presented with shortness of breath, fever, chills and cough with purulent sputum production for the last 2 days. He is conscious coherent, BP :105/70 mmHg, Pulse : 110 beats per min, breathing rate 32 breaths per min, and temperature 38.9 °C. Bronchial breath sounds are heard on auscultation of the right chest. Blood tests show elevated WBC count , urea 9 mMol/L, creatinine 1.6 mg% sodium 142 mmol/l and oxygen saturation (room air) 92%. A chest radiograph demonstrates moderate cardiomegaly and a right lower lobe infiltrate with air bronchograms. Which one of the following is the appropriate management decision for this patient?

a. Treat as an outpatient, start empirical antibiotic therapy without further examinations

b. Treat as an outpatient, take blood and sputum cultures, start empirical antibiotic therapy

c. Admit to hospital, start empirical antibiotic therapy within 4 h of admission

d. Admit to ICU, take blood and sputum cultures and Gram stains, start antibiotic therapy according to results

Consolidation spreading rapidly, causing lobar expansion and bulging of the adjacent fissure inferiorly .

C/F= Red Jelly Sputum

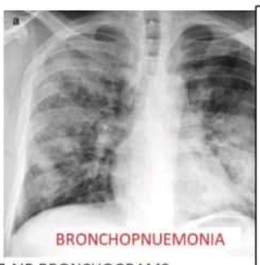
Diagnosis Klebsiella pneumoniae involving the right upper lobe .

Table 1. CURB-65 Scoring

Clinical Feature	Points
Confusion (defined as a Mental Test Score of ≤8, or disorientation in person, place, or time)	1
Uremia: blood urea >7 mmol/L (~19 mg/dL)	1
Respiratory rate: ≥30 breaths/minute	1
Blood pressure: systolic <90 mm Hg or diastolic ≤60 mm Hg	1
Age ≥65 years	1
Total points	

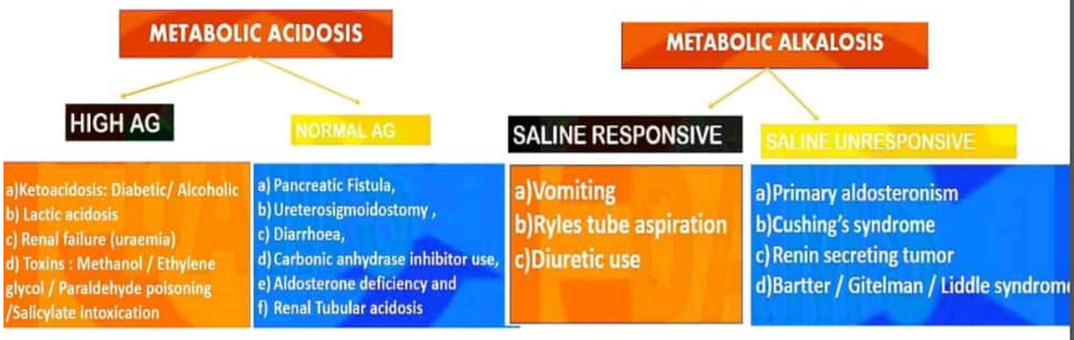
Table 2. Treatment Options Based on CURB-65 Score

Score	Group	Treatment Options
0 or 1	Group 1; mortality low (1.5%)	Low risk; consider home treatment
2	Group 2; mortality intermediate (9.2%)	Consider hospital-supervised treatment (either short-stay inpatient or hospital-supervised outpatient)
≥3	Group 3; mortality high (22%)	Manage in hospital as severe pneumonia; consider admission to intensive care unit, especially with CURB-65 score of 4 or 5



BOTH SHOW PRESENCE OF AIR BRONCHOGRAMS

MC typical pneumonia: strept. pneumonia
MC of atypical pneumonia: Mycoplasma
MC noscomial pneumonia : gram negative bacilli
MC early VAP : Streptococcus
MC late VAP : pseudomonas
MC VAP : pseudomonas
MC pneumonia in cystic fibrosis : pseudomonas
MC pneumonia resulting in ICU admission : streptococcus
MCC pneumonia in HIV : tuberculosis
MC trigger for asthma exacerbation : Rhinovirus
MC cause exacerbation of COPD : Bacterial (H. influenza / strept.)



COVID – 19

- Causative agent: SARS- CoV-2 (Severe Acute Respiratory Syndrome causing Coronavirus II'
- Risk Factors for development of ARDS:
  - MOST IMPORTANT: Elevated D-dimer levels (Indicates Bad prognosis)
  - Lymphopenia

BLOOD Investigations

High D-dimer levels and more severe lymphopenia have been associated with mortality

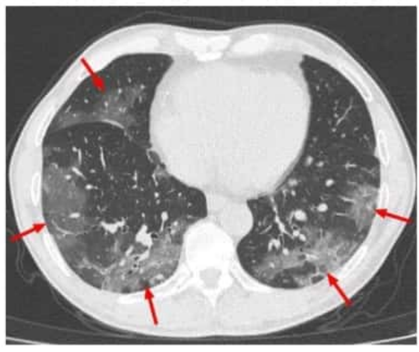
Investigations	Trend
WBC	N → Mild leucocytosis
Lymphocytes	Low
CRP	Elevated
LDH	Elevated
Troponin	Elevated
D dimer	Elevated
Ferritin	Elevated
AST ALT	Elevated
Creatinine Kinase	Elevated

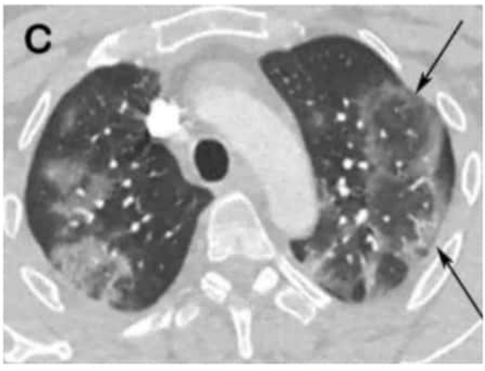
- Fastest method for diagnosis: Rapid Antigen test
- Diagnosis:
  - Testing by Real Time RT-PCR (Gold std result in 4-6 hrs)
  - TrueNAT (result in 1-2 hrs)
  - Rapid antigen kits (15 -20 min)
- CT CHEST is not diagnostic of COVID-19, only useful for estimating the degree of suspicion.
- CT CHEST typically shows
  - Peripheral, B/L Ground glass Opacities with or without consolidation or 'crazy paving'
  - Multifocal ground glass opacities of rounded morphology with or without consolidation or 'crazy paving'
  - Reverse Halo sign

- Indeterminate appearances are absence of typical findings but presence of:
  - Multifocal, diffuse, perihilar or unilateral ground glass opacities with or without consolidation lacking a specific distribution and are noon-rounded or non-peripheral.
- Atypical findings: Absence of typical/indeterminate findings with presence of:
  - Isolated lobar/segmental consolidation without ground glass opacities
  - Discrete small nodules (centrilobular, 'tree in bud')
  - Lung cavitation
  - Smooth interlobular septal thickening with pleural effusion

COVID-19

Typical findings	Atypical findings
Multifocal groundglass opacities	Central or peribronchovascular
Peripheral and basal distribution	More apical distribution
Unsharp demarcation	Lymphadenopathy *
Vascular thickening	
Round	**Very Atypical**
Crazy paving	Cavitation - calcification
Ground glass and Consolidations	Tree-in-bud, bronchiolitis
(Reversed) halo	Nodular pattern
Spider web	Mass
	Pleural thickening





CO-RADS*
Level of suspicion COVID-19 infection

		CT findings
CO-RADS 1	No	normal or non-infectious abnormalities
CO-RADS 2	Low	abnormalities consistent with infections other than COVID-19
CO-RADS 3	Indeterminate	unclear whether COVID-19 is present
CO-RADS 4	High	abnormalities suspicious for COVID-19
CO-RADS 5	Very high	typical COVID-19
CO-RADS 6	PCR +	

NOTES NEXT DOOR

HEALTHY IMMUNE RESPONSE
- Virus inactivated by antibodies or infected cells cleared
- Minimal inflammation and lung damage

HYPER INFLAMMATORY RESPONSE
- ↑ Infiltration of monocytes , macrophages
- Cytokine storm → Multi organ damage

◦ **Which of the following drug is known to decrease mortality in critically ill COVID pt ?**

a. Remdesivir
b. Hydroxychloroquine
c. Toclizumab
d. Dexamethasone

TREATMENT

- ✓ STEROID
- ✓ IL6 INHIBITORS → TOCILIZUMAB

- ✓ REMDESIVIR: RNA dependent RNA Polymerase
  S/E: Hypersensitivity & Hepato-toxic
- ✓ FAVIPIRAVIR→ Broad Spectrum anti-viral → used for ARDS
- ✓ Lopinavir, Ritonavir, Oseltamivir
- ✓ UMIFENOVIR
- ✓ Chloroquine phosphate → Revoked
- ✓ Hydroxychloroquine
- ✓ Azithromycin
- ✓ Ivermectin
- ✓ Convalescent plasma drugs:
  - BEVACIZUMAB (VGFR inhibitor)
  - SARILUZUMAB (IL6 Inhibitor)
  - TOCILIZUMAB (IL6 Inhibitor)
  - LERONLIMAB (IL6 Inhibitor) – to tackle Cytokine storm
  - Methylprednisolone
  - FINGOLIMOD
  - AVIPTADIL (inhibits pro-inflammatory cytokines)
- Interferon Beta 1

High Flow Nasal Oxygen

Indications of IMV- invasive mechanical ventilation
1. Obstructed/absent breathing
2. Severe respiratory distress
3. Central cyanosis, shock, coma, convulsions

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Clinical Guidance for Management of Covid-19 Suspect / Confirmed Cases

Covid -19 Suspect / Confirmed Case

Stratification on the basis of disease severity

Mild	Moderate	Severe
(Fever / Upper Respiratory Tract Infection)	• Pneumonia with no signs of severe disease • RR ≥ 24 / min, SpO_2 < 94% on room air	Respiratory distress requiring mechanical ventilation (non-invasive & invasive) RR ≥ 30 / min, SpO_2 < 90% on room air
Admit to Covid Care Center (CCC) / Home Isolation	Admit in Dedicated COVID Health Centre (DCHC)	Admit in Dedicated COVID Hospital (DCH)
• Contact and droplet precautions • Strict hand hygiene • Symptomatic management • Tab HCQ (400 mg BD x 1 day f/b 400 mg OD x 4 days) may be considered in patients with high-risk features[1] – preferably after shifting to DCHC or at home under strict medical supervision • **For home isolation patients,** seek medical attention when following warning symptoms/signs occur: ○ Difficulty in breathing ○ Persistent pain/ pressure in the chest ○ Mental confusion or inability to arouse ○ Developing bluish discoloration of lips / face ○ Decreased urine output ○ As advised by treating medical officer	**Oxygen Support** • Target SpO_2: 92-96% (88-92% in patients with COPD) • Preferred device for oxygenation: Non-rebreathing face mask (if HFNC or simple nasal cannula is used, N95 mask should be applied over it) • Awake Proning may be used as a rescue therapy (NIH protocol) • All patients should have daily 12-lead ECG • Follow CRP, D-dimer & Ferritin every 48-72 hourly (if available); CBC with differential count, Absolute Lymphocyte count, KFT/LFT daily • Tab HCQ (400 mg BD x 1 day f/b 400 mg OD x 4 days) after ECG Assessment • Consider IV methylprednisolone 0.5 to 1 mg/kg for 3 days (preferably within 48 hours of admission or if oxygen requirement is increasing) **Anticoagulation** • Prophylactic dose of UFH[2] or LMWH[2] (e.g. enoxaparin 40 mg daily SC) **Monitor for & shift to DCH** if any of the following occurs: • Increased Work of breathing (use of accessary muscles) • Hemodynamic instability • Increase in oxygen requirement	• Cautious trial of CPAP with oro-nasal mask / NIV with helmet interface/HFNC, if work of breathing is low • Maintain euvolemia • Consider IV methylprednisolone 1 to 2 mg/kg per day for 5-7 days (in 2 divided doses), if not given already • High prophylactic dose of UFH or LMWH (e.g. enoxaparin 40 mg or 0.5 mg/kg BD SC) if not at high risk of bleeding[3] • Consider intubation if work of breathing is high / not tolerating NIV[4] **Ventilator management** • Use conventional ARDSnet protocol (LTV, proning, etc.) • If sepsis / septic shock: Manage as per existing protocol and local antibiogram • Use sedation and nutrition therapy as per existing guideline
Testing While attending suspect case as per above protocol based on clinical assessment, testing shall be resorted to & if negative - manage in a non-Covid facility according to clinical diagnosis	**Discharge** After clinical improvement, discharge as per discharge policy	**Investigational Therapies[5]** Remdesivir (EUA), Tocilizumab (Off label) & Convalescent plasma (Off label)

1 **High-risk patients for severe disease** include:
- Age: 60 years or more
- Hypertension, DM (diabetes mellitus) & other immunocompromised states
- Chronic lung/ kidney / liver disease
- Cerebrovascular disease
- Obesity (BMI > 25 kg / m2)

2 **LMWH:** Low Molecular Weight Heparin; if no contraindication or high risk of bleeding; **UFH:** Unfractionated heparin

3 **Risk of bleeding:** Use validated score for assessing bleeding risk (eg HAS-BLED score)

3 Use D-dimer and SIC score for **further risk stratification** (SIC score ≥4 portends high thrombotic risk)

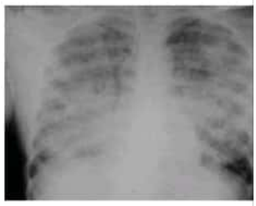
3 Follow AHA/ESC and ISTH guidelines in case patient is **on antiplatelet agents**

4 **Higher chances of NIV failure**

5 Informed and shared decision making is essential before prescribing any of these therapies

NOTES NEXT DOC

Case: A 45/M had COVID pneumonia with spo2 85 % & had to be intubated for respiratory failure. His Neutrophil / Lymphocyte ratio is 7, CRP is 80 ug /mL , ferritin 1000ng /ml & D-dimer : 0.4 ug/ml. The ventilator settings are assist control with tidal volume 500 mL and frequency 15 /min. She weighs 60 kg. His chest xray is given below . True regarding management is :



a. Avoid sedation and neuromuscular blockade as pt is in respiratory failure- **FALSE, AS PATIENT MUST BE SEDATED WHEN INTUBATED**
b. Prone positioning is not indicated as pt is ventilated - **FALSE**
c. Decrease tidal volume to 360 mL. – TRUE DUE TO LOW TIDAL VOLUME MECHANICAL VENTILATION PROTOCOL (4-5ML/KG)
d. Decrease ventilator frequency to 10 breaths per min - **FALSE**

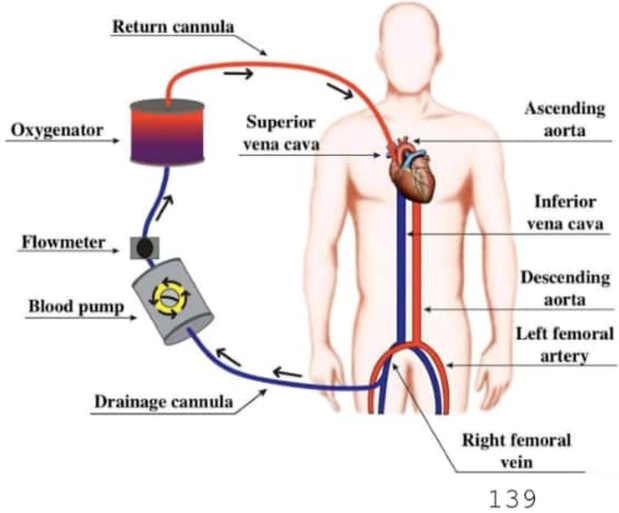
CXR FINDINGS: Cardiac size is normal, B/L Pulmonary edema, no signs of pleural effusion → typical findings of ARDS

Ventilation Strategy: Manage as per ARDS NET protocol

- Low-volume : tidal volume of 6 ml/kg (predicted body weight)
- A plateau airway pressure (Pplat) of ≤ 30 cm H20
- Sedation and Paralytics to relax patient and facilitate ventilation
- Adequate PEEP
- Prone positioning , ECMO can be used for refractory hypoxemia

ECMO-

Extracorporeal Membrane Oxygenation



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